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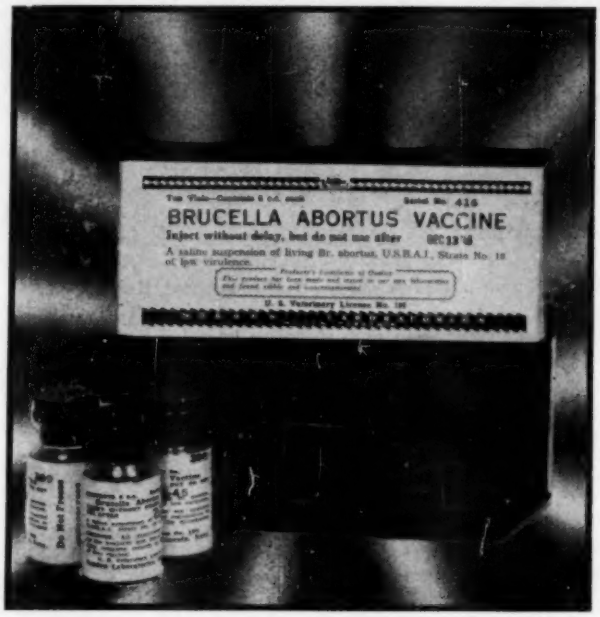

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Lead Poisoning of Cattle

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LEAD POISONING of cattle is infrequently reported. It is believed that many more cases, due to plumbism, occur than are actually diagnosed. Postmortem lesions often are of little or no aid in recognizing the actual cause of death. In many instances, careful autopsies fail to reveal changes that are of any aid in clinically diagnosing the condition correctly. This fact is impressive when the records of the diagnosis laboratory are examined. Quite often, the lesions observed are suggestive of the presence of a bacterial infection. It is hoped that a reminder will serve a useful purpose, because not infrequently, cattle have been vaccinated against disease, when as a matter of fact, the losses in those particular herds were due to the consumption of lead in toxic amounts. We are all familiar with the lesions of hemorrhagic septicemia, but we must remember that a diagnosis of hemorrhagic septicemia cannot be made upon lesions alone. The lesions in lead poisoning and hemorrhagic septicemia are similar. This is true also in other septicemic diseases. Obtaining the case history carefully and subjecting various tissues to a bacteriologic examination are essential in arriving at a correct diagnosis.

The usual source of lead is paint, irrespective of whether the cattle lick it from buildings, pens, yards, tanks, etc. that were painted recently or years before. Empty paint cans used as calf-feeding milk pails, that were not carefully cleaned, are known to have been the source of toxic material.

Paper No. 2237, Scientific Journal Series, Minnesota Agricultural Experiment Station.

From the Division of Veterinary Medicine, University Farm, St. Paul, Minn.

Minnesota has many lakes, and the majority of property owners take pride in their lakeshore cabins which they usually keep well painted. Frequently, the lakeshore lots adjoin cattle pastures into which the discarded paint cans are tossed. The cans are usually found by the cattle, and shortly after they have licked the cans, the cattle become ill. In several known instances, the careless disposal of paint cans has resulted in innocent individuals having to pay damages resulting from losses due to lead poisoning.

Display signs placed in pastures and painted guards on railway tracks have, in several instances, been responsible for the death of a number of cattle. In several instances, painted metal signs used carelessly about farm buildings are known to have caused the death of calves.

Another source of lead poisoning is the carelessly discarded chunk of unusable white lead. Instances have been investigated where brush was piled on top of the white lead; cattle found it, and the brush was actually broken into small pieces by the trampling and milling of the curious cattle. In one instance of this kind, 15 cattle died before the actual cause was determined. In the meantime, the herd had been vaccinated for hemorrhagic septicemia without affecting the progress of the disease.

Judging from observations, one is led to believe that cattle have a desire or craving for lead. Calves of any age have the same inexplainable desire. Our investigations include more cases in calves than in older animals. This may be due to the dangerous

practice of painting calf pens with paint containing white lead. Often, if a careful search is made, it also will be found that recently painted buildings are the source of the trouble.

Other lead combinations of importance in toxicology are lead oxide, red oxide of lead, lead acetate, and arsenate of lead. The latter compound contains arsenic, but its toxic action generally is due to lead. Several instances of lead poisoning have occurred where barrels used for mixing sprays were carelessly left standing in the field. Later in the season, cattle were pastured in the field. The barrels had become dry and had fallen apart; the cattle licked the barrel staves and, as a result, a number of the animals died. A chemical examination was not made, but it was quite evident that lead poisoning was the cause of death. Owners should be warned regarding the danger of grass and hay in fields adjoining orchards or other places where spraying has been done. It is conceivable that spraying trees in a strong wind would be dangerous. Paint sprayers are dangerous where cattle have access to the paint falling on the ground.

Often, it is difficult to obtain a true or complete history of losses from the owners, even on questioning them carefully regarding the possibility of cattle having access to paint or white lead. Usually this is not due to evasiveness but rather to the fact that the inexperienced owner fails to recognize the danger of anything so insignificant as an old discarded paint can. We are satisfied that where the diagnosis is obscure, it is often necessary to investigate the premises personally in order to eliminate the possibility of lead poisoning. A few cases may illustrate the above. A breeder of purebred beef cattle had a number of young calves and yearlings that were found dead in paddocks. The herdsman was questioned as to the possibility of the animals having had access to paint. According to the answers, there was no possible chance. Yet on investigation, it was found that the yards were enclosed by a board fence which had been freshly painted white. Inspection disclosed that the paint had been licked from the surface of many areas of the posts and from the grass at the base of the posts.

In another instance, an owner had lost 6 young calves. The losses occurred during a period of several weeks. Petechial hemorrhages were observed on the heart, the surface of the lungs, and the mucosa of the abomasum. Hemorrhagic enteritis was also present. Diarrhea, alternating with constipation, was observed. Bacteriologic examination gave negative results. The owner was carefully questioned as to the possibility of lead poisoning. We were assured that there was no source from which lead poison could have come. On account of past experience, it was decided to analyze the liver chemically. Lead was found in toxic amounts. On inspection of the premises, it was found that the calf pens were partially constructed with discarded, painted metal signs. The calves had licked most of the paint from the surface.

Symptoms.—The symptoms of lead poisoning as manifested by cattle are not always characteristic. They may, and quite often do, vary. Many of the older practitioners were taught that lead poisoning produces blue lines along the gingivae of the incisor teeth. As most cases are acute, one seldom encounters the blue line which is indicative of chronic lead poisoning. In acute cases, the onset is sudden and death usually follows a short illness. Prostration, staggering gait, or inability to rise are generally observed. A number of instances have been observed where poisoned cattle walked aimlessly about or in circles. Disturbed vision is a prominent symptom; the animals will actually walk into fences or the walls of an enclosure. Frequently, they press their heads against a wall or manger. Grinding the teeth constantly or intermittently is another nervous manifestation. Neck curvature, lateral or dorsal, is common. Epileptic convulsions are infrequently observed. As a rule, the temperature is normal, but it may rise, especially if complications occur.

Usually, there is complete loss of appetite. There may be diarrhea or constipation. Occasionally, diarrhea and constipation may alternate. In cases of diarrhea, the feces often are black and decidedly fetid.

In cases of chronic lead poisoning, the termination may be delayed for several days or more. Since lead is accumulative, it depends upon the amount that is consumed.

Postmortem Findings.— Usually, the lesions found at autopsy are not striking and unless one has a careful history of the

case, an incorrect diagnosis can easily be made. The latter occurs more frequently than it should. An acute hemorrhagic gastroenteritis of the abomasum and small intestine, whenever present, is the most prominent observation. Quite often, the liver has a yellow appearance with marked degeneration. This change also results from other causes. The hepatic changes are not likely to develop in acute lead poisoning. Ecchymotic hemorrhages beneath the capsule of the kidney, extending into the cortex, were observed in one case.

Meningitis may be present. Histopathological examination should demonstrate whether or not one might be dealing with other complications. Such was the case in several of our experiences. Cuffing of the blood vessels was diffusely scattered throughout sections of the cerebrum and cerebellum examined. Bacteriologic studies revealed the presence of an infectious agent, and Listerella organisms were recovered from the brains of 3 cattle. These cases originated from one herd, and chemical analyses demonstrated the presence of lead in toxic amounts in each instance.

In our experience, as in the experiences of others, it has not been possible to make a diagnosis of lead poisoning as a result of histopathological examination of tissues. The lesions as described above conform with those mentioned by other writers.

Chemical Examinations.—It has not been possible to make chemical examinations of the liver in all suspected cases of lead poisoning. Sufficient help has not been available. Positive diagnoses of lead poisoning of cattle have been made without support of a chemical examination, but in those instances, visible evidence of the consumption of lead in some form was present. Analyses are often advisable, especially when a clinical diagnosis is not readily forthcoming. Chemical analyses are also helpful when the owner is doubtful of the clinical diagnosis but will accept the results of a chemical examination.

In several instances, analyses have been made on general principles, that is, where there was a slight possibility that plumbism might be present. Chemical examination in one instance revealed less than 0.3 mg. per cent of lead.

During the past year, 11 cases of lead poisoning occurring in eleven different places have been diagnosed. Diagnosis was accomplished by a careful study of symptoms and history. In the majority of these cases, the owners have been asked to describe the action of the cattle. It was not unusual to hear the owner state that several cattle died over an extended period. In one instance, 6 died during a six-week interval. In the above case, no chemical examinations were made.

TECHNIQUE FOR CHEMICAL EXAMINATION

The lead determinations were made by the dithiazone method of Bambach and Burkey,[1] with minor modifications. Twenty-five grams of wet liver were wet ashed with nitric acid. The ash was extracted several times with small amounts of hot[1] N. HCl and filtered. The filtrate was diluted so that 1 cc. of extract represented 1 Gm. of wet liver. The lead concentration was estimated visually against lead nitrate standards by the dithiazone method.

Table 1 lists the results of the examination for lead by the use of the preceding technique.

TABLE 1—Lead Analysis on Bovine Liver

Case	Milligrams of Lead per 100 Gm. of Liver (wet basis)	Case	Milligrams of Lead per 100 Gm. of Liver (wet basis)
1	2.0	10	Large amount
2	3.2	11	0.3
3	2.3	12	1.0
Control	0.05	13	Negative
4	0.5	14*	2.5
5	1.0	15	0.5
6	0.8	16	0.4
Control	0.8	17	2.5
7	1.5	18	1.7
8	2.0	19	9.0
9	12.0	20	0.25

*This calf appeared normal and died forty-five minutes after drinking milk from a pail; convulsions were observed.

Results of Chemical Examinations.— No experimental feeding trials were conducted. We do not know how much lead of the total intake is absorbed. It is natural to assume that the course of the disease is determined by the amount of intake. It is not to be inferred that case 14 died as the result of a single intake of lead.

Interpretation of the analytical results of the chemical examinations are based entirely upon personal opinions. Concentrations of 0.3 mg. per cent of lead on a wet basis have been considered of no signifi-

[1]Bambach, Karl, and Burkey, Roland E.: Microdetermination of Lead by Dithiazone. Ind. Eng. Chem., Ann. Ed., *14*, (1942): 904.

cance; 0.5 mg. per cent are viewed with considerable suspicion and may be lethal. An amount of 1.0 mg. per cent of lead or more, we consider as positive evidence of lead in lethal amounts.

CONCLUSIONS

It is believed that lead poisoning of cattle and especially of calves occurs more frequently than reports would indicate. It is difficult without a chemical examination to make a positive diagnosis of plumbism in cases where one cannot detect any evidence that lead has been consumed. It should be kept in mind that cattle often find the toxic material even though dumped or discarded in out-of-the-way places. Cattle seem to have an uncanny ability to locate the same. The method used in analysis is not difficult and is not a lengthy procedure.

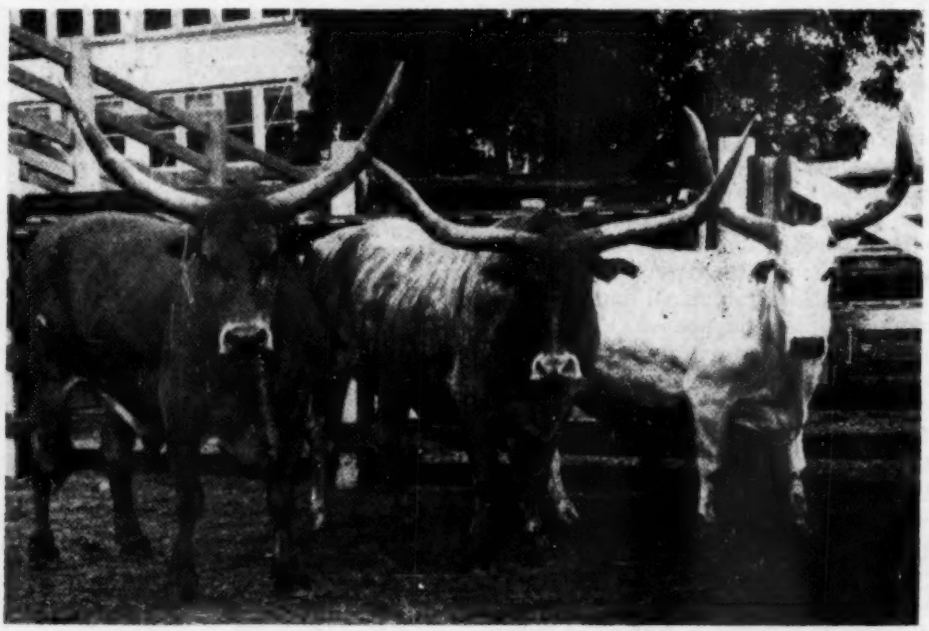
Branding of range cattle causes damage to the hides, which is estimated to cost tanners $6 million worth of leather yearly.

"Eggs Must Again Compete"

With the prospect of abundance, if not surplus, of meat flowing into the food market, "eggs must again compete," says *Poultry Tribune*, which is another way of telling the poultry growers to provide good, clean, tasty eggs. The veterinarian who does engage in poultry practice (some do) is in a strategic position to help in that respect, for the reason that good eggs and good health go hand in hand. The tasty egg is not an accident. It is the product of the well-fed hen cooped in clean, sanitary quarters. Successful poultry practice in the absence of these is not worth bothering with. The poultry farmer, the veterinarian, and the egg eater should agree that "eggs must compete."

During September 1945, 6,582 horses were slaughtered under federal inspection, against 3,711 in that month of 1944.

Longhorns Still Sell



—Courtesy The Cattleman

Three Longhorns were recently sold by L. J. McNeill, of Brazoria, Texas, to the Houston Packing Co. The red steer at the left weighed 1,780 lb., the brindle 1,765 lb., and the white Brahman-cross 1,480 lb. The latter, however, was only 9 years old, while the other two were 11.

Although they rate as curiosities, these steers grazed some of the best pasture in Texas, supporting two cows per acre the year around. The cost of raising them was very little.

Studies of Vesicular Stomatitis with Special Reference to a Virus of Swine Origin

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VESICULAR STOMATITIS (V.S.) has been recognized and specifically identified in the United States on numerous occasions within the past thirty years. In sporadic outbreaks, which have been mostly localized in small areas, horses and cattle have been affected to about an equal extent. Prior to 1943, the disease had not been reported in swine, though these, as well as other animals, had, on numerous occasions, been in intimate contact with affected cattle and horses. Schoening and Crawford[1,2,3] first definitely diagnosed V.S. in North American swine in 1943. In this outbreak, which was confined to a veterinary biological establishment in Missouri, horses, cattle, and goats on the premises remained peculiarly free of clinical evidences of the disease. The object of this paper is to report continued studies of this virus that were undertaken for the purpose of determining its pathogenic qualities and immunologic relationship to previously typed strains of V.S. virus from the equine and bovine species. Other studies, not previously reported, are included.

MATERIALS AND METHODS

In addition to the swine strain which is herein designated (N.J.-M), three other previously typed strains of V.S. virus were employed. A New Jersey type of the virus was represented by a laboratory strain (N.J.) which was originally isolated from a New Jersey cow by Cotton.[4] The Indiana type of the virus was represented by two strains; one a laboratory strain, designated Ind., isolated from an Indiana case by Cotton,[5] and the other (Ind.-C) was obtained in 1942 by one of the writers (A.H.F.) from an equine specimen submitted by E. Heiny, bureau inspector in charge, Denver, Colo. In 1944, Dr. Heiny submitted a number of bovine and equine specimens from Colorado. From these, three strains of V.S. virus were isolated by the writers. All three strains (N.J.-C_1, N.J.-C_2, N.J.-C_3) have been typed as examples of the immunologic group universally referred to as the New Jersey, or N.J. type. An example of such a typing test is given in this paper under the heading, "Immunologic Identification of the Swine Virus." The details of the classifications made in 1944 are not included in this paper. Mention of the isolations is made here as a matter of record and in explanation of the references to these strains that are included in this paper.

Most of the work here reported was conducted with the previously described N.J., N.J.-M, Ind., and Ind.-C strains, which were propagated by serial inoculations in the skin of the hind plantar (metatarsal) pads of guinea pigs and the oral mucosa of large animals. Previous to these studies, the N.J. and Ind. strains had been subjected to a series of intracerebral passages in laboratory and large animals in the course of studies of neuroinvasiveness.[6,7] Lyophilized brain tissue was used in reëstablishing the viruses in epithelial tissue, in which it was carried in series by passages through guinea pigs and large animals. Epithelial tissue, usually freshly harvested but sometimes stored for a few days to as long as ten days in buffered glycerine solution in the refrigerator, was used for all the inoculations reported here.

The inoculum consisted primarily of vesicle covering from lesions on the feet of guinea pigs or swine, the snouts of swine, or the tongues of cattle or horses. This was finely triturated with a sterile mortar and pestle, either with or without the addition of sterile sand. Physiologic salt solution and, in some cases, vesicular fluid were added in varying amounts according to the intended route of inoculation. To inoculum being prepared for local application on epithelial tissue, only sufficient fluid was added to make a thick paste. For intravenous, subcutaneous, or intramuscular inoculation, the diluent was added at the rate of about 1 cc. to each guinea pig pad or an approximately equivalent amount of vesicle covering from large animals. For some of the latter inoculations, the suspensions were filtered through gauze or cotton, or were centrifuged at low speed for five minutes. In most instances, however, the tissue was sufficiently triturated to permit passage of the particles through the needle used for inoculation, and filtration or centrifugation was not employed. The end product for inoculation was held during the inoculation procedures in a receptacle containing ice and water. All subcutaneous, intramuscular and intravenous injections were given in 2-cc. amounts.

The animals used in these tests consisted of 87 cattle, 14 horses, 84 swine, 40 sheep, and over 500 guinea pigs. The cattle, with the exception of 3 suckling calves, were cows which had been used in brucellosis experiments. The horses had not been used previously in experimental studies of other diseases. The swine originated mostly at the Animal

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Disease Station and Animal Husbandry Unit of the Beltsville Research Center, Beltsville, Md., though some were obtained locally in Maryland. The animals, designated pigs or shoats, were young swine weighing 60 to 110 lb.; where the term "hog" was used, it applied to mature animals, usually sows which weighed 200 to more than 700 lb. Most of the swine had previously been immunized against hog cholera (H.C.), except for 30 pigs which were used for production of H.C. virus. The sheep originated at the Research Center. Thirty of them had been previously inoculated with ecthyma virus; 11 had never been used in experimental work. Guinea pigs were obtained from the breeding unit at the Animal Disease Station or from commercial breeders. All animals were classified as normal if previously not exposed to V.S. Those previously exposed to V.S. and recovered were identified as V.S.-immune, the type and strain of virus being indicated by the symbols given above.

Horses and sheep received only local inoculations on the tongue. Guinea pigs received local inoculations on the metatarsal pads. Swine were inoculated locally on the snout or intravenously. Cattle were inoculated locally on the tongue, dental pad, or gum; subcutaneously at the side of the neck; intramuscularly a few inches below the tuber ischii or in a few cases in the neck; or intravenously. In preparing animals for a so-called local inoculation, the site for exposure, *i.e.*, the tongue, dental pad, gum, snout, or metatarsal pad was first scarified (fig. 1, 2, 5). This was done with a sharp knife or a specially constructed instrument composed of a number of Bard-Parker blades, appropriately spaced and fastened together in an assembled unit. A series of linear cuts were made in the dermis or mucosa without inducing marked bleeding which might tend to dilute the inoculum or wash it away. Guinea pigs were at times inoculated by making a number of intradermic tunnels and pricks in the skin of the foot pad with a hypodermic needle.[8] The inoculum was rubbed into the scarifications with the pestle used for triturating the virus, or with a sterile cotton swab on an applicator.

In all inoculation tests involving previously exposed or known immune animals, controls, at least guinea pigs, were provided. All inoculated animals were observed for at least ten days, and, with the exception of a part of the intramuscularly inoculated animals, body temperatures of all large animals were recorded twice and sometimes thrice daily throughout the observation period.

At various times after exposure to V.S. virus, samples of venous blood were collected from some of the large animals. Each sample of blood, whole and unmodified, citrated, or mechanically defibrinated, was inoculated in the metatarsal pads of 2 to 4 guinea pigs for the detection of V.S. virus.

Except for a part of the intramuscularly inoculated cows and some of the guinea pigs, the animals were housed in quarantined, screened concrete-floored, and readily cleansed and disinfected buildings at the Animal Disease Station. Rubber clothing, including boots, coats, and gloves, was worn. A solution of sodium hydroxide, or common lye, in a concentration of at least 2 per cent was used to disinfect this clothing and other contaminated articles. It is appropriate to remark that spontaneous V.S. has never been observed at the Animal Disease Station.

Immunologic Identification of the Swine Virus

The N.J.-M strain of virus, recovered from swine by Schoening and Crawford, was submitted by them to the writers in the form of pad tissue from inoculated guinea pigs. After an additional passage in guinea pigs, the virus was implanted on the tongues of a normal horse and a normal cow, the snouts of 2 normal pigs, and the plantar pads of 4 normal guinea pigs (table 1). All animals developed vesicles typical of V.S. at the site of inoculation in thirty to forty-eight hours (fig. 3, 4, and 6). At the same time, the same virus was inoculated intravenously into a cow and a pig, and intramuscularly into a second pig. None of these 3 animals developed vesicular lesions. The 2 pigs were febrile for several days, possibly due, in part at least, to a Pasteurella organism which was isolated on two occasions from the blood of the intravenously inoculated pig. An Ind.-C-immune cow, 4 guinea pigs immune to Ind. virus, and 1 cow and 4 guinea pigs immune to N.J. virus were also inoculated. The Ind.-immune and Ind.-C-immune animals all developed V.S., whereas none of the N. J.-immune animals were affected.

The horse, 4 pigs, and 4 cows, inoculated as above with the N.J.-M virus, were bled at intervals varying from sixteen hours to five days after inoculation. In all, 20 samples were taken from the 9 animals. The samples were inoculated into the plantar pads of normal guinea pigs for the detection of virus in the blood. None of the guinea pigs developed vesicles.

Fifty-two days after the above intravenous inoculations in the cow and 1 pig, and the intramuscular inoculation in 1 pig, these animals were reëxposed to N.J.-M virus by local inoculations on the tongue or snout. Unexpectedly, the cow developed typical V.S. lesions, though the pigs remained normal. Twenty-nine days later, these 3 animals and all the others which had been inoculated previously with the N.J.-M virus, exclusive of the guinea pigs, were inoculated on the tongue or snout with N.J. virus. In addition, 2 normal shoats

Fig. 1—Scarified area on horse's tongue before application of inoculum.

Fig. 2—Scarified area on gum, dorsal to dental pad of a cow, before application of inoculum.

Fig. 3—Freshly ruptured vesicle on a horse's tongue, forty-eight hours after local inoculation of V.S. virus.

Fig. 4—Large, freshly ruptured vesicle on a cow's tongue, forty-eight hours after local inoculation of V.S. virus.

TABLE I—Immunologic Identification of Vesicular Stomatitis Virus (N.J.-M) From Swine—1943

Species of Animal and No.	Condition of Animal	Method of Inoculation and Results on Indicated Virus: N.J.-M Strain (Inoc. Aug. 21) Method	Result[5]	N.J.-M Strain (Inoc. Oct. 12) Method	Result	N.J. Lab. Strain (Inoc. Nov. 10) Method	Result	Ind. Lab. Strain (Inoc. Nov. 24) Method	Result
Horse 1368	Normal	Lingual	+	....	....	Lingual	—	Lingual	+
Cow 2276	Ind.-C immune[1]	Lingual	+	....	....	Lingual	—	Lingual	+
Cow 2652	N.J. immune[2]	Lingual	+	....	....	Lingual	—	Lingual	—
Cow 2322	Normal	Lingual	—	....	....	Lingual	—	Lingual	+
Cow 2527	Normal	Intravenous	—	Lingual	+	Lingual	—	Lingual	+
Pig 6212	Normal	Intravenous	—	Snout	—	Snout	—	Snout	+
Pig 6205	Normal	Intramuscular	—	Snout	—	Snout	—	Snout	+
Pigs 6206, 6209	Normal	Snout	+	Snout	—	Snout	—	Snout	+
G. pigs 1 to 4	Ind.-C immune[1]	Plantar	+	....	....	....	....	....	....
G. pigs 5 to 8	N.J. immune[2]	Plantar	+	....	....	....	....	....	....
G. pigs 9 to 12	Normal[3]	Plantar	—	....	....	....	....	....	....
Horses 1372, 1374, 1375	Normal[3]	....	....	Lingual	+	....	....	....	....
Cows 2211, 2519, 2261	Normal	....	....	Lingual	+	....	....	....	....
Pigs 6306, 6308, 6314, 6315; Hogs 6171, 6173	Normal[3]	....	....	Snout	+	....	....	....	....
Pigs 6324, 6326	Normal	....	....	....	...	Snout	+	Snout	+
G. pigs 13 to 18	Normal	....	....	....	....	Plantar	+	....	....
Hogs 1 to 12	N.J.-M immune[4]	....	....	....	....	....	....	Snout	+

[1]Recovered from tongue inoculation ten months before with Indiana type virus (Colorado strain); *see* "Materials and Methods."
[2]Recovered from tongue inoculation eight months before with laboratory strain of New Jersey type virus, (N. J.); *see* "Materials and Methods."
[3]Previously inoculated locally with tissue suspected of containing vesicular stomatitis virus, without resultant lesions.
[4]For details, *see* experiment B. [5]+ Indicates development of typical lesions of vesicular stomatitis;—indicates absence of lesions.

and 6 guinea pigs were inoculated as controls. All controls developed typical V.S. lesions, whereas none of the animals which had previously been inoculated with the N.J.-M virus did so (fig. 7). Two weeks after this inoculation with N.J. virus, all the animals which had been inoculated with either or both of the N.J.-M and N.J. viruses, except the guinea pigs, were inocuated *via* scarifications with Ind.-C virus. Only the Ind-C-immune cow failed to develop typical lesions. The results of these inoculations are summarized in table 1. The N.J.-M strain of virus, derived from swine, was concluded to be immunologically indistinguishable from the N.J. type of virus and distinct from the Ind. type. Additional evidence as to the immunologic character of this virus was obtained in later studies (experiment B).

Pathogenicity of N. J.-M Virus for Swine

Experiment A.—This was set up with the intent of duplicating, as far as possible, the conditions under which V.S. had occurred spontaneously in swine.[3]

A group of 12 sows, averaging 283 lb., was assembled in a pen 10 ft. by 18 ft., with a concrete floor and center floor drain. Although none of the animals was noticeably lame, only 1 was found without contusions, abrasions, or cuts of some degree on one or more feet. From all appearances, the hogs were normal otherwise.

On the next day after the hogs had been penned together, the soles of the feet of 3 of the animals were deeply scarified. All four feet of the hog with sound feet were scarified, and two feet of another hog and one foot of a third were similarly treated. These 3 animals, together with 3 of which the feet were not scarified, constituted an uninoculated group for contacts.

The remaining 6 hogs were inoculated intravenously with N.J.-M virus contained in a suspension of infected guinea pig pads (9th passage). In addition, 4 of the animals received intravenously 150 cc., 250 cc., 350 cc., and 1,800 cc., respectively, of H.C. virus, consisting of pooled, chilled, defibrinated blood from 3 pigs, collected on the sixth day after inoculation. The pigs displayed typical symptoms of H.C., and the lesions found *post mortem* were those of

the typical, uncomplicated, acute disease. The pooled virus was culturally sterile. According to the weights of the 4 hogs given H.C. virus, only 1, that which was given 1,800 cc., received a full hypering dose (5 cc. per pound of body weight), such as is used in the commercial production of anti-hog-cholera serum.

No bedding was provided in the pen where the hogs were held. Occasionally, but not regularly, the floor was flushed off with running water. The animals were confined in a bleeding crate and closely examined individually each day. When necessary, the feet were washed with water and a cloth preparatory for examination.

On the third day after the above inoculations, 1 of the sows that had been inoculated with V.S. virus alone was found to have developed vesicles on one foot (fig. 8). On the fourth and fifth days, 3 more inoculated animals, including the 2 which had received the smaller quantities of H.C. virus, and 1 which had received V.S. virus alone, had similar foot lesions. On the fifth day, or two days after lesions had been first observed on the inoculated animals, lesions were found on 4 of the contact hogs. In the remaining 2 contacts, lesions were not discovered until two days later. All 12 animals, except the one which had been given a complete "hypering" dose of H.C. virus, developed fever and lesions on the feet or nose by the seventh day. Vesicles were not detected on the excepted animal until the twelfth day. One foot and the snout were involved at that time.

In from one to four days after the first lesions appeared, additional lesions occurred on the feet or noses of 8 animals. Snout lesions developed in 5 of the animals. Of the 6 contact animals, 4 developed secondary lesions. These involved one foot in 3 cases, and three feet and the nose in 1 case. Three of the 12 hogs in the test eventually developed vesicles on all four feet; 4 developed lesions on three feet; 3 on two feet; and, in the 2 animals which received the larger quantities of H.C. virus in addition to V.S. virus, only one foot was involved. There was otherwise no apparent significant difference in the extent or number of lesions in the inoculated and contact animals, nor was there any evidence that scarification had promoted contact infection. Body temperatures exceeding 104 F. and ascending to 105.6 and 107.4 F. were observed just prior to development of lesions and for a varying time thereafter in the entire group of swine. The fever, which was generally undulant and somewhat intermittent in character, appeared in the inoculated animals as early as the second day and persisted as long as the fifteenth day after inoculation. Individuals in the contact group became febrile only after four to six days, and the fever continued through the eleventh day.

TABLE 2—Experiment A. Results of Intravenous and Contact Exposure of Hog-Cholera-Immune and Hog-Cholera-Hyperimmunized Sows to M Strain of New Jersey (N.J.) Type of Vesicular Stomatitis Virus

Hog	Treatment	Quantity Virus Inoculated (cc.)	Method Of Exposure	Location of Lesions in Hogs[1] and Results of Inoculations of Blood in Guinea pigs[2], in Indicated Number of Days[3]								
				3	4	5	6	7	8	9	11	12
6039	M strain N. J. virus	2	Intrav.	RF	RH	LH	.....	S	....	....	....	.....
5932	M strain N. J. virus	2	Intrav.	....	....	RF	LF, LH	RH	....	....	....	.....
5917	M strain N. J. virus	2	Intrav.	....	....	RH	.....	S, LH, RF	....	....	....	.....
	Hog-cholera virus	150										
5908	M strain N. J. virus	2	Intrav.	....	RH	.....	.....	LH, LF	....	....	....	.....
	Hog-cholera virus	250										
6047	M strain N. J. virus	2	Intrav.	....	....	.....	.....	LH	....	....	....	.....
	Hog-cholera virus	350										
6056	M strain N. J. virus	2	Intrav.	....	....	.....	.....	.....	....	....	....	S, LF
	Hog-cholera virus	1800										
6051	None	None	Contact	....	....	.....	B—	RF, LH, B—	B—	....	B—	.....
5933	None	None	Contact	....	....	RH, B—	.....	LH, B—	B+	....	B—	.....
6041	None	None	Contact	....	....	S, LH, RH, LF, B—	.....	RF, B—	B—	B+	B—	.....
5938	Scarification, LF	None	Contact	....	....	B—	.....	LF, LH, B—	B—	....	B—	.....
6048	Scarification, LF, LH, RH	None	Contact	....	....	RF, LH, B—	.....	RH, B—	B—	B—	B—	.....
5990	Scarification, RF, LF, RH, LH	None	Contact	....	....	RH, B—	.....	S, LF, RF, LH, B—	B—	B—	B—	.....

[1] RF, right front foot; LF, left front foot; RH, right hind foot; LH, left hind foot; S, snout.
[2] B—, negative; and B+, positive results.
[3] Observations were made also on the first, second, and tenth days, but as no changes (....) were noted and bleedings were not made, the data are omitted.
The M strain of N. J. virus, alone and in combination with hog-cholera virus, produced typical lesions of vesicular stomatitis in control guinea pigs.

The lesions on the feet caused varying degrees of lameness, and, during the febrile period, feed consumption was materially decreased. One sow with lesions on all four feet was down and could be gotten on her feet only with difficulty, for a period of nine days. However, she, as well as the others, eventually recovered. But, in all cases, healing was slow and characteristic evidences of past infection remained for several weeks to months (fig. 9 and 10).

Between the fifth and eleventh days after inoculation of the 6 hogs in this test, 27 blood samples were secured from the other 6 contact hogs, and each sample was inoculated on the foot pads of 2 to 4 guinea pigs. Samples from 2 of the hogs, collected on the third and fourth days after the appearance of primary vesicles, produced typical lesions in the inoculated guinea pigs. All other samples failed to produce vesicles, and the guinea pigs in these groups were found to be susceptible when reëxposed several months later to known active virus of homologous type. Experiment A is summarized in table 2.

Experiment B.— Twenty-six days after the V.S. virus had been inoculated in experiment A and fourteen days after the last hog developed lesions, the 12 sows were moved to a similar pen in another barn where 2 normal pigs and 2 normal hogs were placed with them as contacts. At the same time, a similar group of normal animals was placed in the uncleaned pen from which the 12 affected hogs had been removed. One of the hogs that was penned with the 12 convalescent animals was injured and became paralyzed, and, on the ninth day, this animal was replaced by another normal hog. With this exception, these swine remained in their respective pens for twenty-nine days, and none developed lesions of V.S. On the twenty-ninth day, the 4 swine which had been in contact with the 12 sows were removed and placed with the 4 animals in the original uncleaned pen. This group of 8 swine, 1 normal hog, and 1 normal pig were then inoculated on the snouts with N.J.-M virus (first passage, in horses and cows, of virus from swine in experiment A). Within forty-

TABLE 3—Experiment C, Results of Intravenous and Contact Exposure of Hog-Cholera-Immune and Hog-Cholera Hyperimmunized Hogs[1] to M Strain of New Jersey (N.J.) Type Vesicular Stomatitis Virus

Pen	Hog No.	Treatment	Method	Quantity Inoculated (cc.)	Results of Exposure to Virus, Including Location of Lesions[2] in Indicated Number of Days[4] After Exposure						
					4	5	7	9	28	31[5]	34[6]
A	5744	M strain N.J. virus[3]	Intrav.	2	—	—	—	—	....	—	—
		Hog-cholera virus		2325							
	5745	M strain N.J. virus	Intrav.	2	dead						
		Hog-cholera virus		2350							
	5740	M strain N.J. virus	Intrav.	2	—	RF, LF,	RH	....	Killed		
		Hog-cholera virus		2610		LH, S					
B	5743	M strain N.J. virus	Intrav.	2	—	—	S, LH	LF	Killed		
		Hog-cholera virus		2800	—	—					
	5746	None	Contact	None	—	—	—	—	....	—	—
	5738	None	Contact	None	—	—	—	—	....	—	—
	5739	M strain N.J. virus	Intrav.	2	—	S, RF, RH	LF	....	Killed		
	6042	M strain N.J. virus	Intrav.	2	—	S, LH, RH	....	....	Killed		
C	5742	Hog-cholera virus	Intrav.	2450	—	—	—	—	....	—	—
	5747	Hog-cholera virus	Intrav.	2725	—	—	—	—	....	—	—
	5822	None	Contact	None	—	—	—	—	....	—	—
	6016	None	Contact	None	—	—	—	—	....	—	—

[1]All animals hog-cholera-immune.

[2]Five guina pigs inoculated on pads as controls, developed typical lesions.

[3]RF, right front foot; LF, left front foot; RH, right hind foot; LH, left hind foot; S, snout; —, no lesions; ...., no change.

[4]Observations made also on first, second, third, sixth, and eighth to twelfth days, but as no changes (....) occurred, data are omitted.

[5]Animals reëxposed on twenty-ninth day by snout inoculation with M strain of virus. Only observations on second day are recorded. Observations were continued, however, through the tenth day after inoculation. Three guinea pigs inoculated as controls developed typical takes.

[6]Again exposed on thirty-second day by snout inoculation with another sample of M strain of virus. Observations on second day only are recorded, though daily examinations were continued through the tenth day after inoculation. Two pigs inoculated on the snout and 5 guinea pigs inoculated on the pads with the same virus, as controls: all developed typical takes.

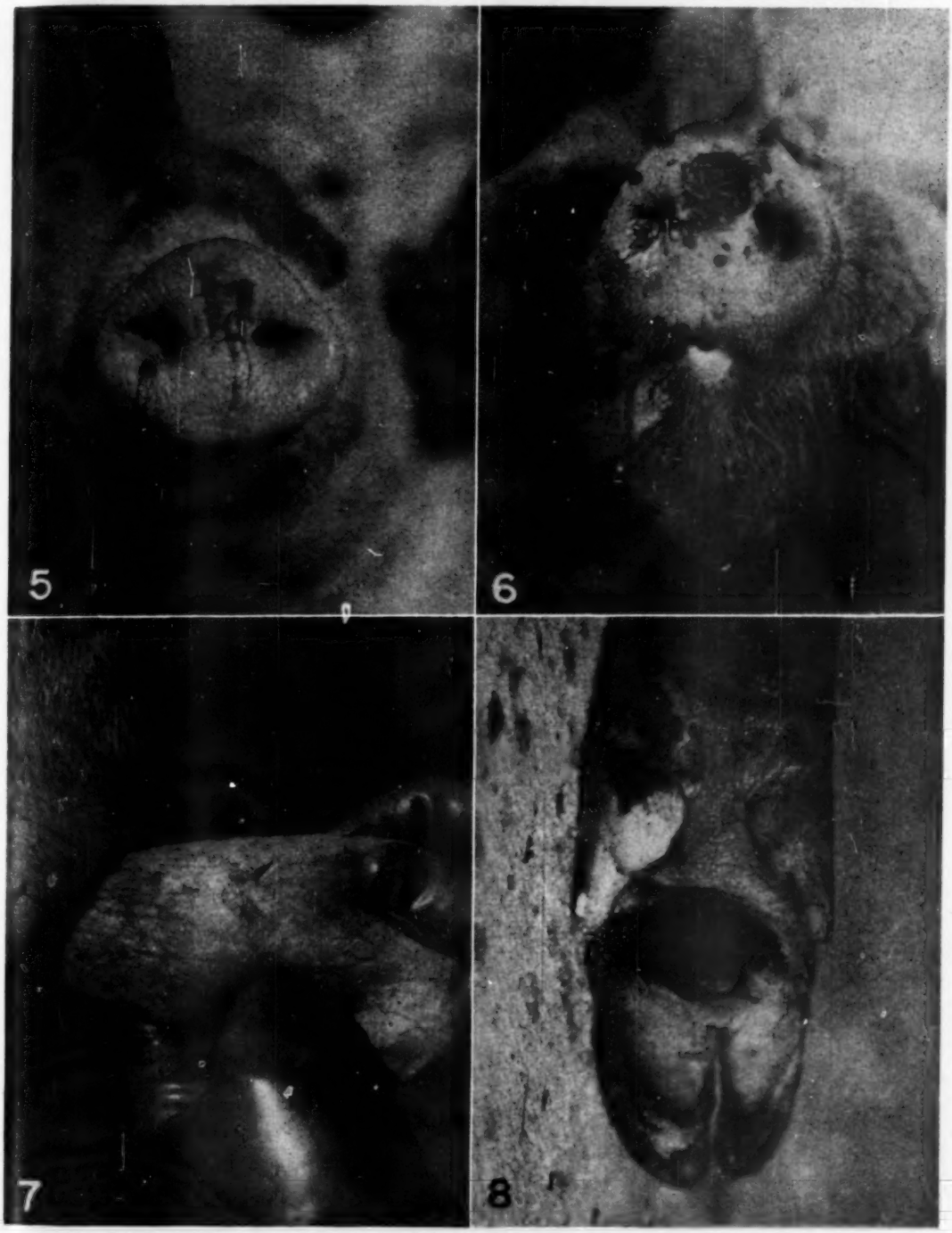

Fig. 5—Scarified area on pig's snout before application of inoculum.

Fig. 6—Ruptured vesicles and erosions on snout and in nostril of a pig, forty-eight hours after local inoculation of V.S. virus.

Fig. 7—Site of inoculation of V.S. virus on immune horse's tongue, forty-eight hours after inoculation.

Fig. 8—Large, freshly ruptured vesicle at the heel and vesicle on the dew claw of a hog, following intravenous inoculation of V.S. virus.

TABLE 4—Results of Intramuscular Inoculation and Subsequent Lingual Inoculation of Cattle[1] with Vesicular Stomatitis Virus[2]

Group	Cattle No. Head	Status	Date	Virus[3]	Method[4]	Result[5]	Date	Virus	Method	Result	Date	Virus	Method	Result	Date	Virus	Method	Result
1	10	Principals	11/10/43	N.J.	Intram. or Subcut.	—	12/6/43	N.J.	Ling.	—	....	....	....	....	....	....	....	....
	1	Control	....	....	....	....	12/6/43	N.J.	Ling.	+	....	....		....		....	....	....
2	8	Principals	1/21/44	NJ-M	Intram.	—	2/21/44	NJ-M	Ling.	—	3/4/44	Ind.	Intram.	—	4/8/44	Ind.-C	Ling.	—
	2	Principals	1/21/44	NJ-M	Intram.	—	2/21/44	NJ-M	Ling.	+	3/4/44	Ind.	Intram.	—	4/8/44	Ind.-C	Ling.	—
	1	Principals	....	....	....	....	....	....	....	....	3/4/44	Ind.	Intram.	—	4/8/44	Ind.-C	Ling.	—
	1	Control	....	....	....	....	2/21/44	NJ-M	Ling.	+	....	....	....	....	....	....	....	....
	2	Control	....	....	....	....	....	....	....	....	....	....	....	....	4/8/44	Ind.-C	Ling.	+
3	23	Principals	3/30/44	NJ-M	Intram.	—	4/10/44	Ind.-C	Intram.	—	4/20/44	NJ-M	Ling.	—	5/1/44	Ind.-C	Ling.	—
	1	Principals	3/30/44	NJ-M	Intram.	—	4/10/44	Ind.-C	Intram.	—	4/20/44	NJ-M	Ling.	—	(died)	....	....	....
	2	Principals	....	....	....	....	4/10/44	Ind.-C	Intram.	—	....	....	....	....	5/1/44	Ind.-C	Ling.	—
	1	Control	....	....	....	....	....	....	....	....	4/20/44	NJ-M	Ling.	+	....	....	....	....
	1	Control	....	....	....	....	....	....	....	....	....	....	....	....	5/1/44	Ind.-C	Ling.	—[6]
4	13	Principals	5/17/44	Ind.-C	Intram.	—	5/27/44	NJ-M	Intram.	—	6/8/44	Ind.-C	Ling.	—	6/17/44	NJ-M	Ling.	—
	3	Principals	....	....	....	....	5/27/44	NJ-M	Intram.	—	6/8/44	Ind.-C	Ling.	—	6/17/44	NJ-M	Ling.	—
	1	Control	....	....	....	....	....	....	....	....	6/8/44	Ind.-C	Ling.	+	....	....	....	....
	1	Control	....	....	....	....	....	....	....	....	....	....	....	....	6/17/44	NJ-M	Ling.	+

[1]All cows, except 2 of 10 principals in group 1, which were calves.

[2]Virus used for intramuscular inoculations of each group was controlled in guinea pigs. Typical vesicular lesions developed in each case.

[3]Groups 1 and 2: guinea pig passage virus; groups 3 and 4: cow passage virus. N.J. laboratory strain New Jersey type virus; N.J.-M, swine strain of New Jersey type virus; Ind., laboratory strain of Indiana type virus; Ind.-C, Colorado strain of Indiana type virus.

[4]Intram., intramuscular; Subcut., subcutaneous; Ling., lingual.

[5]—, No lesions of vesicular stomatitis; +, lesions of vesicular stomatitis.

[6]Guinea pigs inoculated with the same virus at the same time, developed lesions of vesicular stomatitis, but only after ninety-six hours.

eight hours, all 10 animals had developed typical vesicles at the site of inoculation. These spread peripherally and finally involved the entire surface of the snout in most cases. On the third day after the appearance of snout lesions, secondary vesicles were present on one or more feet of 7 of the 10 animals. The 3 swine which did not develop secondary lesions on the feet were in different categories: One had been exposed to the recovering sows; 1 had been held in the original uncleaned pen; and 1 was the recently introduced control pig.

The 12 original sows from experiment A were next exposed by inoculation on the snout with N.J. virus, fifty-five days after their original exposure to N.J.-M virus. All animals remained normal. Two control pigs exposed at the same time developed vesicular lesions. Fourteen days after this exposure, the above 14 swine were reinoculated on the snout with Ind. virus. At the same time, the 4 pigs used for immunologic identification test were similarly inoculated (table 1). All developed typical, spreading, vesicular lesions on the snout, but no secondary lesions appeared on the feet.

Experiment C.— This experiment was next conducted for the purpose of obtaining more information on the pathogenesis of the N.J.-M strain of V.S. virus, especially in relation to hyperimmunization with H.C. virus. In consideration of the possibility that the daily close examinations of the feet of the animals in experiment A, especially the washing that had been frequently resorted to, might have been a factor in the infection of uninoculated contacts, it was decided to forego these procedures, in so far as was practical, in experiment C. Fever and lameness were accepted as primary indicators of incipient or existent vesicles, and the feet and snouts were observed with as little direct handling as possible.

Twelve H.C.-immune sows averaging 532 lb. were obtained from the breeding stock of the Animal Disease Station. Two sows were placed in pen A and 4 in pen B, both in the same barn, and 6 were placed in pen C in another barn.

The H.C. virus was obtained from 27 pigs that were bled out on the sixth day after inoculation for the purpose of this test. The animals had displayed a typical H.C. syndrome, and lesions of the typical, acute, uncomplicated disease were found on postmortem examination. *Salmonella choleraesuis* was demonstrated in the defibrinated, pooled blood.

The 2 sows in pen A were inoculated intravenously with 2 cc. of N.J.-M V.S. virus (second passage of swine virus from experiment B in guinea pigs). This was followed by a full "hypering" dose of H.C. virus (5 cc. per pound of body weight). Two of the 4 sows in pen B were inoculated like the 2 in pen A, and 2 were left as uninoculated contacts. Of the third group of 6 sows in pen C, 2 were inoculated intravenously with V.S. virus alone, 2 were inoculated intravenously with a complete "hypering" dose of H.C. virus alone, and 2 were left as uninoculated contacts.

Owing to the cold weather prevailing at the time of this experiment, it was considered desirable to keep the pens well bedded with dry straw. The pens were not cleaned during the course of the test. Examinations were made for twelve consecutive days. Blood samples were drawn daily from the 2 contact hogs in pen C for a period of ten days, beginning on the second day after their pen mates had been inoculated. The 20 samples were inoculated individually on the plantar pads of 3 guinea pigs. Neither the contact sows nor the guinea pigs inoculated with their blood developed lesions of V.S.

Both sows in pen A developed a septicemia as a result of the intravenous inoculations. The animals ate sparingly; their skin became highly congested, and 1 of them went down and died on the fourth day after inoculation. The second sow recovered after an illness lasting a few days. Neither animal developed vesicular lesions.

In pen B, both inoculated sows developed vesicles, whereas neither of the 2 contact animals did.

In pen C, only the V.S.-inoculated hogs developed lesions of the disease.

Latent lesions occurred in 3 of the 4 V.S.-inoculated sows in pens B and C. These 4 animals were removed from the pens and slaughtered twenty-eight days after the beginning of the test. At this time, residual lesions were present on the feet (fig. 9). The claws were removed from five of the affected feet, and scrapings were made from the depths of the laminar tissue. This material, in three composite lots, was tri-

turated with physiologic salt solution and inoculated into the plantar pads of guinea pigs without producing lesions of V.S.

On the twenty-ninth day after the beginning of experiment C, the 7 remaining animals which had failed to develop lesions of V.S. were reëxposed to N.J.-M virus (second passage, in guinea pigs, of swine virus from experiment B) by snout inoculation. Exposed at the same time was a previously uninoculated and presumably susceptible sow which had been isolated in a corner pen of the barn where experiment A had been conducted. Guinea pigs inoculated as controls developed typical V.S. lesions, but none of the 8 sows did. Three days later, the hogs were again exposed by inoculations on the snout with N.J.-M virus which had been harvested from a cow's tongue (animal exposed with second guinea pig passage of virus from experiment B), and stored in buffered glycerine in the refrigerator for a period of ten days. Two hogs from the same original source as the 8, but which had never contacted V.S. in any way, were included as controls. Only the 2 controls developed lesions; all of the 8 reëxposed sows again proved refractory. When the 2 control hogs were slaughtered on the tenth day after exposure, both were found to have developed secondary vesicular lesions on one foot. Experiment C is summarized in table 3.

INTRAMUSCULAR INOCULATIONS OF CATTLE WITH V.S. VIRUS

Olitsky, Traum, and Schoening, in reporting comparative studies of V.S. and foot-and-mouth disease (F. & M.),[8,9] cited the failure of 14 cattle which had been intramuscularly injected by them with V.S. virus to develop the disease. This result was stated by these authors to be in direct contrast to the results obtained by them and others when the virus of F. & M. was injected intramuscularly. Schoening and Crawford[3] inoculated 2 cattle and 4 swine intramuscularly with N.J.-M virus, with negative results. They again suggested that intramuscular inoculation was applicable in the differentiation of the viruses of F. & M. and V.S. The writers have made 110 intramuscular inoculations of 63 cattle with various strains of V.S. virus in an endeavor to obtain more information on this important matter. In addition, the immunity of the intramuscularly inoculated animals was challenged by later inoculations on the tongue.

Eight cows and 2 suckling calves were first injected either intramuscularly or subcutaneously with N.J. virus and all failed to develop vesicular lesions therefrom. Guinea pigs inoculated with the virus on the pads developed typical takes. Twenty-six days later, 1 normal cow was added to the group and the 11 cattle were inoculated on the tongue with N.J. virus. The normal cow developed lesions within 48 hours, but all the 10 previously inoculated animals remained normal (table 4, group 1).

A second group of 10 cows was inoculated intramuscularly with N.J.-M virus, from which no vesicular lesions resulted. Guinea pigs inoculated on the pads as controls developed V.S. A month later the intramuscularly inoculated cows and a normal control cow were exposed by lingual inoculation with virus of the same strain. Eight of the cows remained normal, while the control and 2 of the intramuscularly inoculated cows developed typical lesions of V.S. in the usual time. Twelve days after the last inoculation, the entire group was injected intramuscularly with Ind. virus. All animals remained free of V.S. lesions, while guinea pigs inoculated as controls developed the disease. Thirty-five days later, the 11 cows and 2 normal control cows were inoculated on the tongues with Ind.-C virus. Only the control animals developed lesions of V.S. (table 4, group 2).

The inocula used in the previously described intramuscular tests were prepared from guinea pig pad tissue. In view of the fact that most differential diagnoses would probably involve bovine tissue, and in consideration of the possibility that tissue from that species might prove to be more highly infective when inoculated intramuscularly, virus-infected bovine mucosa was used for all subsequent intramuscular inoculations (groups 3 and 4, table 4). The infectivity of each preparation of virus that was injected intramuscularly was checked by plantar inoculation of guinea pigs.

In the course of this work, a total of 40 additional cows (groups 3 and 4, table 4) were inoculated intramuscularly with N.J.-M virus, and, at other times, 39 of these received Ind.-C virus, also intramus-

Fig. 9—Break in integument at coronary band, loosened soles of both claws, and exfoliation and cicatrization of the bulb, on foot of a hog, four weeks after exposure to V.S. virus.

Fig. 10—Hog's foot, three and one-half months after lesions of V.S. first appeared. Note black line of demarcation between old hoof and descending new growth on right claw. Hairless, thickened skin remains on knee as result of continued kneeling in acute stages.

cularly. Again, none of the animals developed V.S. When the immunity of 38 of these animals (1 died before lingual inoculation) was challenged by inoculation of first one and then the other virus on the tongue, all withstood the test. In one instance, however, in which a cow and guinea pigs had been inoculated as controls of the challenge virus, the cow failed to come down, and lesions occurred in the guinea pigs only after ninety-six hours. The cow developed typical lesions when subsequently reëxposed, and the virus which she received in the first instance must be concluded to have been of low virulence. There was, therefore, no thorough check of the immunity of the 25 cows (1 died in the interim) in this test which had been pre-

viously exposed to Ind.-C virus. In all the remaining 84 inoculations, a wholly adequate test of immunity is considered to have been made.

INOCULATIONS OF SHEEP WITH V.S. VIRUS

In all the outbreaks of V.S. which have been reported, the disease has not been observed to affect sheep, although these animals have in numerous instances been in contact with clinically affected animals of other species, *viz.*, horses or cattle. Cotton[4] reported unsuccessful attempts to infect sheep by tongue inoculation. Wagener,[10] on the other hand, reported artificial infection of both sheep and goats with either the Ind. or the N.J. types of virus. Two goats inoculated on the tongue by Schoening and Crawford[3] failed to develop the disease. In view of these contrasting results, the writers inoculated 38 sheep on the scarified tongue with N.J.-M virus, which produced the typical disease in both control cattle and swine. None of the sheep developed typical V.S. Minor ulcerative lesions developed in 2 sheep, but material from these failed to produce V.S. in guinea pigs inoculated with it. Later, 30 of the same sheep were inoculated lingually with Ind.-C virus, which was at the same time implanted on a horse's tongue. The horse developed typical V.S., but the sheep remained without lesions. Two sheep were inoculated lingually with one of the N.J. type viruses isolated in 1944 from Colorado specimens, without producing the disease. The writers have not yet inoculated goats with V.S. virus.

DISCUSSION

It is generally agreed that the lesions of vesicular stomatitis (V.S.), vesicular exanthema (V.E.),[11] and foot-and-mouth disease (F. & M.), considered individually in given affected animals, are essentially indistinguishable. There are differences in contagiousness, epizoötiology, and susceptibility of different species to various methods of exposure with the three viruses, but when any vesicular disease appears in the mouths or feet of ruminants or swine, exact immediate diagnosis is of the greatest importance in order that foot-and-mouth disease may not be overlooked.

The viruses of F. & M., V.S., and V.E. differ in particle size,[12 to 14] and cultivability in tissue cultures and chicken embryos.[15-19] V.S. virus possesses neurotropic potentialities which are apparently lacking in F. & M. and V.E. viruses. Under certain conditions, specific differentiation by means of filtration or serologic methods may be practicable, but animal inoculation tests are generally relied upon for distinguishing these viruses under average conditions. Such tests involve horses, cattle, swine, and guinea pigs, primarily.[3,8,11] Other species are sometimes appropriately included. The resistance of sheep to inoculations of V.S. virus indicates that those animals may well be included among the species used for differentiation between that disease and F. & M.

Intramuscular inoculation of cattle now appears to have been adequately confirmed as a reliable index in the differentiation of the viruses of F. & M. and V.S. Clinically evident V.S. was conspicuously absent in all of the 110 intramuscular inoculations carried out by the writers. The negative results are in complete conformity with the findings of others. In contrast, intramuscular inoculation of the virus of F. & M. in cattle has been reported to produce that disease regularly.[8] The writers have not made intramuscular or other inoculations with F. & M. virus, in conformity with the established policy of prohibition of experimentation with the disease in the United States. This country has been free from the malady for long periods, and on each of the few occasions when the disease has appeared here, it has been successfully eradicated. Every practicable preventive measure, of which strict abstinence from experimentation with the virus is one, should be continued in effect in this country. It would seem appropriate, however, that comprehensive studies of intramuscular inoculations with the various types of F. & M. virus be carried out in countries where experimentation is permissible.

In the tests summarized in table 4, all but 2 of 63 cattle inoculated intramuscularly or subcutaneously with V.S. virus withstood lingual exposure to virus of homologous type twenty to thirty days later. An additional cow, inoculated intramuscularly with virus N.J.-C_2, one of the strains isolated in 1944 from Colorado specimens, developed lesions when inoculated on the tongue seventeen days later. Following conclusion of the in-

oculations summarized in table 4, 6 cattle, 1 from group 2, and 5 from group 4, were again exposed lingually to first one and then the other of the two types of V.S. virus. One of the 6 cattle developed lesions following exposure to Ind. type virus, and 3 others developed lesions following inoculations of N.J. type virus. These results indicate that although a substantial immunity usually follows intramuscular inoculation of V.S. virus, the immunity gained may be of short duration in some animals. That the same may apply to intravenous inoculation is indicated by the failure of a cow to withstand tongue inoculation forty-two days after having been injected intravenously with the same strain of virus (table 1).

The immunity resulting from takes following local inoculations of horses, cows, and guinea pigs on the tongues or foot pads has been considered generally to be long lasting.

In the course of the studies here reported and in connection with immunologic tests conducted for the purpose of classifying the three strains of V.S. virus isolated in 1944, there has been an opportunity for re-exposure of recovered animals to determine the duration of immunity. Seventeen reinoculations have been made on the tongues of 11 horses, at intervals of seventeen days (1 case) to fourteen months (2 cases) after primary artificial infections due to virus of homologous type. All the animals proved to be refractory. Thirty-two similar reinoculations have been made in 19 cows, at intervals varying from eight days (2 cases) to fourteen months (2 cases) after primary infections. At four, twelve, and fourteen months after primary local infections had occurred, 3 animals again developed the disease when reinoculated with the same strain of virus or virus of homologous type. Only about 50 per cent of a large number of guinea pigs reëxposed eight to twelve months after primary infection were found to be fully resistant.

It is apparent, therefore, that in conducting typing tests of V.S. virus, the immunity of recovered animals, especially cattle and guinea pigs, should be checked before proceeding with cross-immunity inoculations. The necessity was fully impressed on the writers in the course of typing tests of the V.S. viruses isolated from Colorado specimens in 1944. All three strains (N.J.-C_1, N.J.-C_2 and N.J.-C_3) readily infected normal horses, cattle, swine, and guinea pigs. Two strains (N.J.-C_1 and N.J.-C_3) were promptly classified as N.J. type by their production of lesions in Ind.-immune animals and the absence of lesions in N.J. immunes. One strain (N.J.-C_2) reacted similarly in type-specific immune horses and guinea pigs, but 2 cattle presumed to be immune to N.J. type virus by reason of previous infection with that virus also developed lesions. Other animals known by reason of a challenge inoculation to be immune to N.J. type virus withstood inoculation of the N.J.-C_2 virus, which was thus eventually concluded to be of the N.J. type.

When challenging the immunity of recovered animals in preparation for their utilization in typing tests, it is well to so inoculate them that there will be a minimum of tissue destruction at sites destined for future inoculation. Cattle may be inoculated on the dental pad or gum, thus leaving the sound tongue for the typing test. In guinea pigs, the integument of the metatarsal pads, which are preferable for diagnostic use, may be preserved by making the challenge inoculations on the pads of the forefeet. In the few cases where the method was tried, inoculation in the gingival tissue of horses failed to produce clinically typical infection. Inoculation on the tongue four days later proved the animals to be susceptible. Possibly, intramuscular, subcutaneous, or intravenous inoculations might be found adaptable for bolstering the immunity of these animals in preparation for reinoculation of virus to be typed. It is noted, however, that breaks in the immunity of horses have not been experienced, when the animals were reëxposed at intervals comparable with those applying in similar tests in cattle.

In the course of the V.S. studies concerned in this paper, the writers have produced primary infections on the lingual mucosa of 27 cows and 14 horses. Only 1 of these animals, a cow, developed secondary lesions following the primary take. In this case, a secondary lesion appeared on one forefoot. Virus was recovered from the lesion and identified immunologically in guinea pigs. Among 26 swine infected by contact or local inoculation on the snout with N.J. type virus, secondary lesions occurred on the feet in 13 animals (50%). No secondary lesions developed in any of

the 18 swine similarly inoculated with Ind. type virus. The incidence of secondary V.S. lesions in pad-inoculated guinea pigs varied from less than 10 per cent with some strains of virus to 50 per cent or more with others. The interest of these observations is in the comparative infrequency of secondary lesions in V.S. in contrast with their reported common occurrence in F. & M. "Secondaries" are also said to be quite common in V.E. of swine.

Experiments by others have shown that V.S. is transmissible by contact between infected and susceptible cattle[4, 5, 8] or swine[10]. In the writers' experiment A, all of 6 contact hogs contracted the disease. In this test, the hogs were kept on a bare, concrete floor, simulating the conditions where an outbreak in swine occurred[3]. In addition, sound feet of 3 of the 6 animals were scarified. In the course of making close daily examinations, the feet were washed with a cloth. In experiment C, 4 contact hogs failed to develop lesions, though all became immune. In this test, the animals were provided with straw bedding, and their feet were not scarified or handled directly except when necessary for proof of the existence of lesions. Either or all of these procedures may have been factors in the different results of the two tests. However, the addition of bedding may have been the principal factor. The U. S. Department of Agriculture Foot-and-Mouth Disease Commission was unsuccessful in producing infection in swine by contact in bedded pens[8]. Cotton[5] concluded that direct contact in the early stages of the disease was necessary for transmitting the infection in cattle. The same appears to be the case with swine. The swine in experiment A were removed to a clean pen thirteen days after the last clinical case developed. Normal swine (experiment B) were then placed with the convalescent animals and in the uncleaned pen from which the latter had been removed. These normal animals contracted neither clinical infection nor immunity.

Cotton[5] demonstrated V.S. virus in the blood of both horses and cattle at intervals between forty-four and sixty-six hours after artificial exposure. Virus was demonstrated by the writers in the blood of 2 contact-infected sows twenty-four and forty-eight hours after the last secondary lesions appeared.

That pigs are less susceptible than adult swine to contact infection with V.S. was suggested by the results obtained by the writers in the course of studies of one of the strains of virus isolated in 1944. Two pigs and 2 sows were placed in a small pen with an inoculated cow which developed typical lesions in forty-eight hours. None of the 4 swine contracted the disease, but the 2 hogs were later found, when inoculated, to be immune, presumably as the result of inapparent or occult infection, as in the case of experiment C with contact hogs. The 2 pigs in contact with the infected cow developed neither clinically apparent infection nor immunity. Again when typing the strain of virus derived from swine, a pig was inoculated intravenously (table 1). Lesions failed to appear in this young animal, whereas they never failed to occur from similar inoculation in mature swine, except in some cases hyperimmunized at the same time with hog-cholera virus. These developments conform with experience in the outbreak at the serum plant, where the disease was almost entirely confined to large hogs.[3]

In inoculations of cattle with V.S. virus, comparatively few immature animals have been used in the Bureau's studies. However, a number of calves have failed to develop lesions when inoculated with virus proved to be infective for adults. Wagener[10] encountered calves which failed to develop lesions following inoculation of virus which had apparently been checked in a horse. In Cotton's studies,[21] a heifer was not infected with virus shown to be virulent for guinea pigs. Wide variations in the virulence of a given strain of V.S. virus may be experienced in the course of experimental passages. It nevertheless appears that until more detailed information is available, the results obtained from inoculations of immature cattle and possibly swine should be interpreted with caution.

SUMMARY AND CONCLUSIONS

The first strain of vesicular stomatitis to be isolated from a natural outbreak in swine has been studied experimentally to determine its pathogenic qualities and immunologic relationship to previously typed strains of vesicular stomatitis virus from cattle and horses. Similar to other isolated strains of vesicular stomatitis virus, this strain was pathogenic for locally exposed horses, cattle, swine, and guinea pigs. This

strain of vesicular stomatitis virus, originally isolated from Missouri swine, has been identified to be of the New Jersey type. Three strains of virus from cases in Colorado cattle and horses proved to be of the same immunologic type. There were 87 cattle, 14 horses, 84 swine, 4 sheep, and over 500 guinea pigs used for this work.

Hog-cholera-immune swine inoculated intravenously with vesicular stomatitis virus readily contracted the latter disease. When the virus was inoculated at the same time with large doses of hog-cholera virus, there was a tendency toward delay or absence of formation of vesicular lesions.

Swine readily contract vesicular stomatitis through contact with affected animals under certain conditions. Contact with convalescent swine or pens from which convalescent swine had been recently removed failed to convey the infection. Occult, or inapparent, infection, resulting in immunity, may result from contact with swine in the acute stages of the disease. Pigs appear to be less susceptible to infection by contact than older swine. Virus was demonstrated in the blood of clinically affected swine which became infected through contact.

Secondary lesions occurred in 13 of 26 swine primarily infected by contact or through inoculation on the snout with New Jersey type virus. Of 18 swine similarly infected with Indiana type virus, none developed "secondaries".

Sheep failed to develop vesicular stomatitis when inoculated with either Indiana or New Jersey type virus. Cattle failed to develop vesicular lesions when inoculated intramuscularly with either type of vesicular stomatitis virus. Some but not all of such cattle became immune. It is suggested that the contrasting results from lingual inoculations of sheep and intramuscular inoculations of cattle with the viruses of foot-and-mouth disease and vesicular stomatitis may be properly applied as an integral part of differential diagnosis of the two diseases.

Either inapparent or clinically evident vesicular stomatitis infections generally produce substantial immunity to the same immunologic type of virus. In some cases, however, measurable resistance is of short duration. In consequence, it becomes necessary to check the immunity of animals that have recovered from type specific infection before proceeding with tests to determine the immunologic character of a virus of unclassified type.

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An Outbreak of Salmonellosis in Horses and Mules

MAJOR DONALD R. CORDY, V.C., and CAPT. ROBERT W. DAVIS, V.C.

BECAUSE of the apparent infrequency of salmonellosis in mature horses and mules, this outbreak of the disease in military animals is reported. *Salmonella abortivo-equinus*[1] and occasionally *Salmonella typhi-murium* and *Salmonella aertrycke*[2,3] have been incriminated in septicemia, joint ill, and colitis of foals. However, enzoötics with high mortality have occurred infrequently in adult horses and mules in Europe and the United States, particularly when resistance has been lowered by rail or sea transport.[4] It is thought that sporadic and enzoötic cases may occur with much greater frequency than is usually believed, and that many clinically diagnosed cases of enteritis and forage poisoning may be salmonellosis.

EPIZOÖTIOLOGY

This outbreak occurred in India during the dry season in the middle of November, 1944, in a group of nearly 300 horses and mules belonging to an American quartermaster pack unit. There was an approximately equal number of horses and mules and mares and geldings, ranging in age from 5 to 15 years. The mules had been shipped directly from the United States; the horses had come from the South Pacific. There was a sketchy history of enteritis among military animals in the latter place at some prior date.

From the Indian port of debarkation the animals had made a rail trip of about one week, without delay in route. After arrival, the group had been placed in an old corral a few miles from the railhead. The fence enclosed a tract of jungly riverbottom about a quarter of a square mile in area, including a stream with a number of sluggish backwaters. The corral had been used off and on for eighteen months for animals of the Chinese Army. Upon occupation of the area, the animals were fed from the ground with barley, gram, and rather poor Indian hay.

Three weeks after arrival the first case appeared. During a period of seven days a total of 8 cases was observed. Seven animals (2 mules and 5 horses) died or were destroyed *in extremis*, and 1 horse recovered. Only the latter animal received sulfaguanidine treatment. Untreated fatal cases showed symptoms for periods of two to thirty-six hours, usually less than twenty-four hours, before death. The surviving case was sick about twenty-four hours before the five-day sulfaguanidine treatment was begun and was clinically recovered at the completion of the treatment.

SYMPTOMATOLOGY

Symptoms shown were fairly uniform throughout the group. Most cases showed anxiety and distress. One case that terminated fatally displayed symptoms similar to those observed in the somnolent type of encephalomyelitis, such as depression, stupor, and leaning the head against the stall. All animals showed marked weakness, and those which lived longer showed posterior incoördination. Many went down and made feeble and unsuccessful attempts to rise. Temperatures were elevated, usually to 103.0-105.6 F. Conjunctivitis was noted in 4 animals. Pharyngeal paralysis was not seen. Pulse and respiration were somewhat accelerated. Diarrhea was irregular in occurrence and never prominent.

GROSS LESIONS

Five cases (2 mules and 3 horses) were examined shortly after death. All had died of the disease, except 1 horse which was destroyed *in extremis* after thirty-six hours of illness.

Severe gastroenteritis was present in all 5 animals. The 2 mules, sick only a few hours, showed more inflammation of the stomach than of the intestine. In the horses, 2 of which were sick about thirty-six hours, the most severe lesions were in the cecum and colon. The earliest cases showed marked hyperemia, some edema, and scattered petechial and ecchymotic hemorrhages in the mucosa. Cases of greater duration showed patchy areas of grayish-red super-

At the time this manuscript was prepared, the authors were serving overseas in the Veterinary Corps.

ficial necrosis in the mucosa of the cecum and colon.

The spleens were slightly swollen or normal in size, and usually the pulp was somewhat softened and dark red. These splenic changes were most marked in the peracute cases in the 2 mules.

The livers were lighter colored than normal, somewhat soft, slightly enlarged, and greasy on the cut surfaces.

Visceral lymph nodes were reddish, juicy, and often hemorrhagic.

Lesions in other organs were less uniform. Small hemorrhages were seen in the endo-

—Photomicrograph by Major J. L. Arbogast, M. C.

Fig. 1—Colon, showing necrotic surface and infiltrated mucosa. (Taken at 440x; prints enlarged 3x exact slide; photographic print ratio unknown.)

cardium of the right atrioventricular valves in 1 animal. Mucosal petechiae occurred in the bladder in 1 case. Hyperemia and some edema of the mucosa were present in this and another bladder. In 1 case, there were petechiae in the kidney cortex. The only brain examined was that of the "sleepy" case. Grossly, the only abnormality was a few petechiae in the walls of the lateral ventricles.

—Photomicrograph by Major J. L. Arbogast, M. C.

Fig. 2—Liver, showing extreme fatty change. (Taken at 440x; prints enlarged 3x exact slide; photographic print ratio unknown.)

HISTOPATHOLOGY

Microscopically, the intestine showed marked hyperemia, small hemorrhages, marked mononuclear infiltration of the mucosa and about the vessels of the adjacent muscularis, and superficial coagulation necrosis of the mucosa over large areas.

The splenic sinuses and pulp were engorged with blood, and actual hemorrhage may have occurred in one of the more acute cases. There was no evidence of any marked proliferation of mononuclear elements.

The hepatic cells contained large fat droplets. Nearly all liver cells were affected, but in a few areas it could be seen that the lesion was initially centrolobular. The liver was markedly hyperemic, and showed accumulations of mononuclear cells in the interlobular tissues. No hepatic necrosis was noted.

Kidney sections from 1 case showed marked hyperemia, small cortical and medullary hemorrhages, and some cloudy swelling of the convoluted tubules. Another case showed hyperemia, a few interstitial mononuclear foci, tubular fatty degeneration, and a few small areas of tubular necrosis.

Section of the brain from the 1 case showed one small hemorrhage in the cerebral cortex. There was no evidence of other brain damage, the cerebrum, cerebellum, and brain stem all having been examined.

ETIOLOGY

A pooled specimen of intestinal contents from the 2 mules which died the first day of the outbreak was rushed to the laboratory. No toxic metals were found. Suggestive colonies developed on Salmonella-Shigella agar. Three colonies were picked and put through differential mediums. All gave similar results. The organism was a small, aërobic, gram-negative, motile rod Glucose.

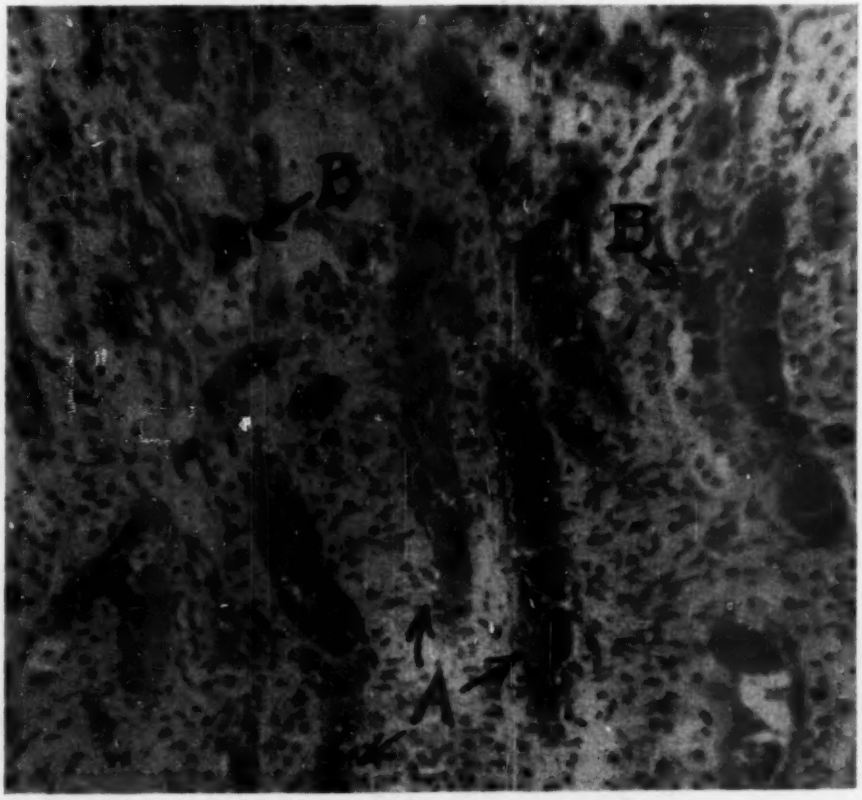
maltose, arabinose, xylose, rhamnose, mannitol, and dulcitol were fermented with formation of acid and gas. Lactose, sucrose, and raffinose were not attacked. Hydrogen-sulfide was produced. Gelatin was not liquefied. Indol was not formed. Nitrates were reduced to nitrites. No growth occurred on Simmons citrate medium. Acid was formed in phenol red tartrate agar.

An identical organism was isolated from the intestinal contents and urine of the third case, a horse.

These strains were all typed serologically by First Lieut. C. D. Cox, Sn.C., as having the antigenic structure (VI, VIII...; r; 1, 3, 5...). This identifies the organism as *Salmonella morbificans* (Bergey *et al.*) which is a member of group C, according to the Kauffmann-White scheme as conducted by Edwards and Brunner.[5] This species was originally isolated from a case of septicemia in a cow, and subsequently from cases of gastroenteritis in man.[6]

TREATMENT

The illness was too brief in most cases for much of any treatment to be instituted. As soon as the first deaths occurred, bunkers were built, and the animals were no longer fed off the ground. After the 2 mules died the first day, all sick animals were evacuated to a nearby remount dispensary. Three died here so rapidly that treatment was hardly begun. A fourth lived thirty-six hours but was destroyed because moribund. The "sleepy" case was supported in a sling for twenty-four hours but died. By this time, the laboratory diagnosis of salmonellosis was definite, so the remaining sick horse was put on sulfaguanidine treatment. At 6 p. m., 600 gr. were given by capsule, and at 10 p. m., 600 gr. more were administered. During the next five days, 250 gr. were given three times daily. Upon completion of this treatment the animal was regarded as clinically recovered. The pack unit, having had no more new cases, had



—Photomicrograph by Major J. L. Arbogast, M. C.

Fig. 3—Kidney, showing (a) engorged vessels, (b) necrotic foci. (Taken at 440x; prints enlarged 3x exact slide; photographic print ratio unknown.)

departed, during this period of treatment, on a hundred-mile road march lasting five days. When visited at the end of this march, no further cases of this type were observed.

DISCUSSION AND CONCLUSIONS

The initial focus of this outbreak could not be determined. The corral may have been contaminated by previously infected animals, or the South Pacific horses, with the history of enteritis in that area, may have included carriers. No opportunity was presented for a fecal survey of the group for carriers.

A specimen of river water from the corral showed algae, bacteria, and much débris, but no enteric pathogens were isolated. A quantity of the worst of the hay issued was fed to healthy animals without ill effects.

It seems very likely that the long rail trip may have been an important predisposing factor.

The incubation period presumably could have been anything up to three weeks, as the source of the infection could not be determined.

Because of the extremely rapid course, distinct symptoms of diagnostic value did not appear. The consistent observation of anxiety, weakness, wobbly gait, going down, and fever are suggestive.

While blood and organs were not cultured, the finding of the organisms in the urine taken at autopsy strongly suggests septicemia rather than the action of blood-borne bacterial toxins from a strictly enteric focus of infection. The brevity of the course would support this supposition.

While all cases were not examined bacteriologically, nor were Koch's postulates completely fulfilled, there seems little doubt of the etiologic relationship of *S. morbificans*.

Routine sanitary control measures instituted by Capt. Kenneth I. Gumaer, V.C., presumably helped greatly to limit the extent of the outbreak. The apparent success of sulfaguanidine in a single case is promising but not conclusive. Because of the probable septicemic nature of the disease, it would appear reasonable to try the more easily absorbed members of the sulfonamide group.

Shortly after this outbreak, a remount unit in another part of India reported six deaths in mules which showed symptoms and gross lesions very similar to those reported herein. These deaths occurred in some 300 mules from another South Pacific area. Unfortunately, no cultural or histopathologic work was done. A fecal survey which was made on 43 mules from the group, a few weeks after the losses, showed no enteric bacterial pathogens. This absence of carriers in a random sample proves nothing but is reported as general information.

SUMMARY

1) An outbreak of eight cases of salmonellosis among 300 horses and mules, after sea and rail shipment, is reported.

2) Weakness, posterior incoördination, and fever were the most consistent symptoms.

3) Severe gastroenteritis with hemorrhage and superficial necrosis of the gut mucosa was the most marked lesion. The cecum and colon were chiefly affected; the liver showed extreme fatty changes, and the spleen was engorged with blood.

4) *Salmonella morbificans* was isolated from the intestinal contents and urine.

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Baby pig disease is less serious when the pigs have the benefit of heat and protection from an electric brooder.

Rabies can be controlled, says Schlotthauer (*Iowa Vet.*, July, 1945) by reducing the opportunity for spread, and by increasing resistance through vaccination. To reduce spread: eliminate stray dogs, confine suspects, quarantine infected areas, and keep licensed dogs under observation (on leash).

Inspection of Human Food

Outline for the Preparation of Governing Legislation

(Prepared by the Special Committee on Food Hygiene)

[At the annual meeting of the Executive Board in August, 1945, a motion was passed requesting the AVMA Special Committee on Food Hygiene to draft a basic food inspection plan for the guidance of interested agencies and individuals. The Committee has drafted the following outline which has been approved for publication by the Executive Board.

The Committee is also preparing a suggested code covering the salient features contained in the outline. This code will be published in a forthcoming issue of the JOURNAL.

Reprints of the outline as here published, and, later on, of the code, will be available upon request to the AVMA office.—The Editors]

The outline here presented is suitable for use by states, counties, municipalities, and other political subdivisions. It provides for the protection of the public health by preventing the use in trade channels of food that is diseased, unsound, unwholesome, or otherwise unfit for human consumption, and is designed also to prevent the misbranding and adulteration of foods. It provides further for the proper disposal of unfit and misbranded foods. It provides for establishing local agencies that can act in coöperation with livestock and public health organizations in the control of diseases of animals and man, thus safeguarding the livestock industry and promoting the public welfare.

The Special Committee on Food Hygiene of the American Veterinary Medical Association has concluded that these purposes can best be accomplished by the organization of a coördinated food-control program under veterinary planning and direction. An outline of the salient features that should be included in proposed legislation follows.

To effect this purpose the legislation should include:

1) **Title**—A statement of the purpose of the enactment.

2) **Definitions**—Definitions clearly setting out: (*a*) The office responsible for the inspection; (*b*) definition of the term "meat" (to include, with proper designation, the flesh of all food animals); (*c*) definitions of other foods; (*d*) definitions of other special terms used in the enactment.

3) **Administration**—Provision for the appointment of a qualified veterinarian in charge to enforce this enactment, with authority to appoint such assistants as he may deem necessary.

4) **Licenses**—A clear statement of the significance of licensing provisions and a statement of the procedures involved in granting licenses upon application and the circumstances under which licenses may be revoked.

5) **Plant Construction and Equipment**—A statement in general language, broad enough to permit the office responsible for inspection to meet changing conditions, prescribing the required type of construction of plants and the character and installation of equipment and necessary facilities for handling products, and for conduct of the inspection with the more important features mentioned specifically, showing the requirements for maintaining clean premises.

6) **Antemortem Inspection**—A requirement for antemortem inspection by veterinarians on the day of slaughtering, stating in general the facilities for this purpose which the plant operator must provide, and including in general language broad principles to guide veterinary inspectors in disposing of animals showing deviations from the normal.

7) **Postmortem Inspection**—Provision for thorough postmortem inspection, at the time of slaughter, by veterinarians, stating generally the requirements which the plant operator must meet to present carcasses and parts for inspection and to handle dressing operations properly, and giving in general language principles governing the disposal of diseased carcasses and parts, which will serve to guide veterinary inspectors.

8) **Time of Operations**—Provision for the veterinarian in charge to designate the hours of the day and the days of the week during which plants may be operated when few animals are slaughtered or when but a small quantity of products is prepared.

9) **Preparation and Handling**—Provision for adequate inspection of meat and other foods

during their preparation and handling, whether in the packing plant, wholesale distributing plant, retail market, restaurant, or other food-handling establishment.

10) **Inspection Legend**—Provision for marking inspected and passed meat and products with a specified inspection legend with a number identifying the plant, this to be applied in a prescribed form whether by branding on the product or printing on labels.

11) **Marking and Labeling**—Provision for adequate labeling (and adequate marking of unlabeled product) to prevent deception and to inform the purchaser as to the common or usual name of the product, the ingredients with which it was prepared, the name and address of the manufacturer, packer, or distributor, and an accurate statement of the quantity of contents in terms of weight, measure, or numerical count, whichever is appropriate. In order to make the label control effective, it is necessary also that provision be made for control of the composition of products prepared with two or more ingredients to insure preparation with proper ingredients and distribution under commonly understood names.

12) **All Products to Be Inspected**—Provision for inspection of all products, within the jurisdiction, in accordance with this enactment, except that inspections by other agencies acceptable to the veterinarian in charge will be recognized.

13) **Access to Premises**—Provision for access by inspectors at any time to all parts of premises covered by the enactment.

14) **Seizure**—Provision for seizure of product wherever found, within the jurisdiction, in the channels of trade, when the inspector has reasonable cause to believe that the product is unfit for food, adulterated, or misbranded.

15) **Disposal of Retained or Seized Products**—Authority for inspectors summarily to dispose of retained or seized products, and to condemn, and to require under the supervision of an inspector the destruction for food purposes of diseased animals, carcasses, parts of carcasses, and unfit or adulterated products, and to require that misbranded products be made to conform to the requirements of this enactment.

16) **Appeals**—Provision for appeal from the decision of an inspector to his immediate superior having jurisdiction over the subject matter of the appeal.

17) **Rules and Regulations**—Authority for the inspection agency to promulgate rules, regulations, and orders implementing the broad terms of the enactment and consistent with it.

18) **Financing**—Provision for financing the inspections required by this enactment through the permanent establishment of an adequate, annual appropriation.

19) **Reports**—Provision for reporting of inspections by inspectors, and the furnishing of information for that purpose by plant operators and owners of products inspected.

20) **Coöperation with Other Agencies**—Provision for the veterinarian in charge to exchange information with public health and other disease-control agencies.

21) **Penalties**—Penalties for failure to comply with any portion of the enactment or the rules, regulations, and orders properly issued thereunder.

22) **Saving Clause**—A provision whereby the invalidation of any section will not affect the legality of the remainder of the enactment.

SPECIAL COMMITTEE ON FOOD HYGIENE

s/ O. W. SEHER, *Chairman*

M. O. BARNES	M. R. CLARKSON
E. M. LYNN	H. E. KINGMAN

Collaborators: H. L. Foust, *Chairman*, Special Committee on Postwar Planning and Wilbur C. Kilpatrick, Field Supervisor, Washington State Division of Dairy and Livestock.

Chicken and Turkey Feathers

The USDA, coöperating with chemists of the United States Rubber Company, announces that 100 million pounds of chicken and turkey feathers will soon be processed into a fabric that will be a strong competitor of wool. The new fabric, softer, lighter, and warmer than wool, will dye any color and will not shrink by washing. It will be produced commercially in 1946, the October, 1945, issue of *U. S. Egg and Poultry Magazine* reports.

Decline in World's Milk Supply

During the last ten years (*Certified Milk*, Oct. 1945), there has been a substantial decline in milk production except in Britain, the United States, and Canada. In the rest of the world, the decline has brought the total below the normal average. The decline was marked in New Zealand, Australia, Ireland, Continental Europe, and Russia. The cause is attributed to a decrease of 12 to 15 per cent in the number of dairy cows, and to shortage of feed, labor, transportation, and dairy equipment.

The government subsidy paid for milk production has made it profitable for dairy farmers to keep their scrub cows, says *Food Industries*.

Experimental Edema and Ascites in Poults

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DURING recent years, the turkey industry has experienced considerable loss in young poults from a condition known variously as ascites, edema, visceral gout, "waterbelly," and other names which possibly are not widely used. The condition is characterized by subcutaneous edema and an accumulation of clear or straw colored fluid in the serous cavities and by rather sudden death following a brief gasping period, apparently brought about by pressure on vital organs and by edema of the lungs.

The condition most frequently attacks poults 5 to 8 days old, and usually disappears from a flock by the end of the second week, although occasionally it extends into the third, fourth, or fifth week. Ordinarily, the history of such flocks includes the information that the birds were doing well before the trouble started. Many also report that such flocks drink excessive amounts of water and that such excessive water intake results in quite fluid droppings.

Symptoms.— Ordinarily, the symptoms are of very brief duration. A husky poult is seen lying down and gasping; the dyspnea becomes progressively worse until the bird dies within a half to one hour. During the latter part of an outbreak, some birds may linger longer. The condition may affect up to 15 or 20 per cent of a flock.

Generally, it is difficult to find live affected birds because symptoms do not develop until a short time before death.

Lesions.—The most striking abnormality found at the autopsy is the accumulation of a clear, serous fluid within the body cavities, particularly the peritoneal. In most instances, this fluid distends the structures of the abdominal wall so tightly as to lead to the term "waterbelly." Hydropericardium, hydrothorax, and edema of the lungs are usually found, and in many instances, the heart is two or three times normal size. In many of the affected birds, varying amounts of white flaky exudate is present in the air sacs, or the exudate may be distributed over the heart, liver, or lungs.

Occasionally, the fluid within the peritoneal cavity will contain considerable fibrin which covers the liver and intestine with a thin whitish layer. We have also occasionally observed hemopericardium.

Many affected birds show a subcutaneous edema of the ventral parts of the body, extending up into the axillary region. The kidneys may be somewhat enlarged, pale, and edematous in some cases.

Cause.—The cause of this condition has been a debated subject. In our experience, numerous attempts to demonstrate an infectious agent through cultural and poult inoculation trials have failed. Correspondence and conversations with eminent poultry pathologists reveal that they likewise do not believe that the condition is caused by an infectious agent. It is true that in some instances it has been possible to isolate organisms of the genus Escherichia or Salmonella. However, these cases are so exceptional that we feel they are only indirectly related to the primary condition.

In 1943, Selye[1] reported the production of nephrosclerosis in baby chicks through the use of a 0.9 per cent solution of sodium chloride as drinking water. His description of postmortem examinations rather closely resembles the condition under consideration.

Delaplane (1934), quoted by Ewing,[2] and Witter[3] produced a "clinical condition resembling visceral gout" in chicks using drinking water containing as low as 0.6 per cent sodium bicarbonate.

In discussing renal insufficiency, MacCollum[4] states: "The specific inability of the glomeruli to excrete water or, more probably, their specific inability to excrete sodium chloride and other inorganic substances may explain the oliguria or anuria of the acuter stages. Sodium chloride passes into the urine through the quite specific activity of some of the epithelial cells, and when these fail, it is retained in the tissues. Since the tissue fluids must remain isotonic for the cells, water is reserved to dilute this concentrated salt solution and the conse-

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quence is edema, hydrothorax, and ascites."

These observations led to the set of experiments reported here, which were set up in an attempt to determine the cause of edema and ascites in poults.

In the conduct of these experiments, 1-day-old poults were used exclusively, and these poults at no time had access to any other feed or water than that listed for each individual pen. A 26.0 per cent protein starting mash containing 0.5 per cent of commercial salt was used as the base, and sodium compounds were in addition to this. This basic mash was one which has been used by a considerable number of raisers without any evidence of it being unsatisfactory in any way. All poults were brooded in electric brooders. Water used was from the city mains unless otherwise specified.

In the first experiment, to find the effect of sodium chloride, 50 poults were divided into five pens of 10 poults each and sodium chloride was administered as shown in table 1. This table also gives the results.

Since the effects of sodium chloride were so marked in this experiment, the second experiment with 10 poults in each pen was set up. Results are shown in table 2.

To determine the effects of sodium bicarbonate administration, experiments 3 and 4 were set up. Ten poults were used in each pen in the experiment for which results are shown in table 3.

In the experiment for which results are reported in table 4, 15 poults were used in each pen.

Since results reported in table 4 were so striking, it was believed desirable to repeat some of the trials using sodium bicarbonate

TABLE 1—Effect of Sodium Chloride

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	10	1.0 per cent added to mash*	One dead ninth day; cause undetermined; no edema or ascites cases; dimissed in twenty-five days.
2	10	1.0 per cent added to drinking water*	All dead in five days; all had extensive edema; 1 with ascites.
3	10	2.0 per cent added to mash*	Four dead in seven days with typical edema and ascites; 6 dismissed in twenty-five days.
4	10	2.0 per cent added to drinking water*	All in stupor in forty-eight hours, and dead in four days; no edema or ascites cases.
5	10	Control	All dismissed as normal in twenty-five days.

*In addition to 0.5 per cent commercial salt already present in the starter mash.

TABLE 2—Effect of Sodium Chloride

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	10	0.5 per cent CP NaCl added to drinking water*	Five dead in sixteen days with varying degrees of edema and ascites; 5 dismissed in twenty-one days.
2	10	0.5 per cent commercial salt added to drinking water*	Eight dead in ten days with varying degrees of edema and ascites; remaining 2 killed after twenty-one days; no lesions.
3	10	Large lumps of commercial salt placed in feeder with mash*	Two dead in nine days with typical edema and ascites; 1 dead, cause undetermined; remainder dismissed in twenty-four days; averaged 6.6 oz. in weight compared with 9 oz. for controls.
4	10	3.0 per cent CP NaCl added to starter mash*	Seven dead with typical edema and ascites in twenty days; 2 dead from omphalitis; remaining 1 had an enlarged heart at autopsy.
5	10	Control	All dismissed as normal after twenty-one days.

*In addition to 0.5 per cent commercial salt already present in the starter mash.

TABLE 3—Effect of Sodium Bicarbonate

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	10	1.0 per cent in starter mash	One dead in nine days with typical edema and ascites; 1 dead in thirteen days, cause undetermined; 8 dismissed in eighteen days.
2	10	3.0 per cent in starter mash	Two dead in six days, of omphalitis; 4 dead with typical edema and ascites in ten days; remaining 4 killed in eighteen days; 2 had enlarged hearts; others normal.
3	10	Control	One dead of injury in five days; remainder dismissed in eighteen days.

solutions as drinking water. The local water supply was rather high in minerals, but the percentage was not definitely known. As a consequence, the experiment reported in table 5 was arranged, using distilled water solutions of sodium bicarbonate as drinking water. Fifteen poults were used in each pen.

To determine with certainty that sodium rather than carbonic acid was responsible for the results reported in tables 4 and 5, the experiment reported in table 6 was arranged. Carbonated water from a local soda fountain was obtained for the purpose. Fifteen poults were used in each pen.

Since both sodium chloride and sodium bicarbonate could be shown to cause edema and ascites and all the other manifestations of this disease, it logically followed that other sodium compounds, if given in the proper dosages, might produce it. Unfortunately, the end of the hatching season was at hand and only odd lots of poults were available. As a consequence, the results of experiments reported in table 7 were, for the most part, uncontrolled. However, it would seem that they are of sufficient interest for inclusion here regardless of this criticism.

In this group of experiments, the various sodium compounds were dissolved in tap water to the extent of 0.75 per cent, and these solutions were used as drinking water.

Failure to produce edema cases with sodium carbonate led to the supposition that the poisonous nature of this chemical

TABLE 4—Effect of Sodium Bicarbonate

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	15	0.025 per cent sodium bicarbonate in drinking water	Two dead of omphalitis in twenty-four hours; remainder dismissed in eighteen days.
2	15	0.025 per cent sodium bicarbonate in drinking water plus 1.0 per cent commercial salt in mash*	Two dead of omphalitis in twenty-four hours; remainder dismissed in eighteen days.
3	15	0.1 per cent sodium bicarbonate in drinking water	One dead of omphalitis in forty-eight hours; remainder dismissed in eighteen days.
4	15	0.3 per cent sodium bicarbonate in drinking water	One dead with unabsorbed yolk in seven days; 3 dead with typical edema and ascites in fifteen days; remainder dismissed in eighteen days.
5	15	0.5 per cent sodium bicarbonate in drinking water	One dead of omphalitis in forty-eight hours; 10 dead with typical edema and ascites in fifteen days; remainder killed in eighteen days; 1 had an enlarged heart; 3 were normal.
6	15	Control	Three dead of omphalitis in forty-eight hours; remainder dismissed in eighteen days.

*In addition to 0.5 per cent commercial salt already present in the starter mash.

is so severe that the dilution used did not permit time for the edematous condition to occur in poults. The same is probably true for sodium iodide.

Only one more opportunity became available for testing sodium carbonate administration to day-old poults, and, in this one, a 0.1 per cent solution was used as drinking water for 18 poults. One typical case of edema with ascites occurred in seven days and another in eleven days. The remaining 16 poults were dismissed as normal in twenty days. It is likely that, in this trial, the dosage was too low to produce numerous cases.

Other experiments were conducted in an effort to determine the cause of this condition.

Two pens of 10 poults each were given 0.1 per cent and 0.5 per cent, respectively, of potassium iodide in starting mash. All birds in each pen were dismissed in twenty days, with no occurrence of the disease.

One pen of 10 poults was given 7.5 per cent of commercial calcium carbonate mixed in the mash. One died of injury and the remainder were dismissed as normal in fifteen days.

In another experiment, water collected from a supply which had been given to a flock in which rather heavy losses had occurred from this condition was concentrated in a desiccator to half its volume and given to a pen of 10 poults for eight days, when the supply was exhausted. Tap water was used for the remainder of the period. No cases had occurred in fifteen days when the birds were dismissed. Appreciation is expressed to Dr. C. D. Lee, of Iowa State College, who was responsible for the concentration of this water.

In an attempt to determine whether or not formaldehyde fumigation would produce the disease, 120 poults which had just recently emerged from the shell were fumigated for one hour in a hatching compartment with 2 oz. of formalin and 1 oz. of potassium permanganate per 268 cubic feet of space. They were then held in a brooder for observation for twenty-six days. Fifty poults not so fumigated served as controls. By the sixteenth day, 6 from the fumigated group had died with edema and ascites, and 2 additional dead ones showed subcutaneous edema only. Twenty-five others from the fumigated pen died over the 26-day period, while 5 of the control group died. None of these losses could be diagnosed as edema or ascites. The birds in the fumigated pen were admittedly crowded after the first week and at least 7 of the nonspecific losses were a result of being trampled. In addition, after the tenth day, when only 2 cases of edema and ascites had occurred, the brooder was purposely allowed to become insanitary in an attempt to enhance any effect which the fumigation may have had. It is believed that these last two factors were probably responsible for the comparatively high death rate in this group.

TABLE 5—Effect of Sodium Bicarbonate

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	15	0.4 per cent sodium bicarbonate in distilled drinking water	Two dead of omphalitis in forty-eight hours; 4 dead with typical edema and ascites in eighteen days; remainder dismissed in twenty-six days.
2	15	0.5 per cent sodium bicarbonate in distilled drinking water	One dead of omphalitis in seventy-two hours; 9 dead with typical edema and ascites in twenty-three days; remainder dismissed in twenty-six days.
3	15	0.6 per cent sodium bicarbonate in distilled drinking water	Three dead of omphalitis in seventy-two hours; 10 dead with typical edema and ascites in eighteen days; 2 remaining killed in twenty-six days; 1 had an enlarged heart; 1 was normal.
4	15	Control Distilled drinking water	One dead of omphalitis in twenty-four hours; 1 dead in twenty-one days, cause undetermined; remainder dismissed in twenty-six days.

DISCUSSION

While it is realized that high sodium intake has not been proved as the cause of field cases of edema and ascites, lesions and anamnestic data are strikingly similar to

those of field cases, and it would appear that there are many sources from which poults might obtain excessive amounts of sodium. Sodium chloride which is added to practically all starter mashes has been shown by many investigators to be necessary for normal development of poultry. Ordinarily, most formulas for starting mashes include from 0.5 to 1.0 per cent of salt. This amount of sodium is in addition to the varying amounts of sodium which the feed already contains, which may be considerable.

The 1939 yearbook of Agriculture, quoted by Ewing,[2] shows high sodium levels particularly for some feeds which are being used as meat-protein substitutes. The list includes corn gluten feed, linseed meal, and cheaper grades of fish meal, all of which contain well over 1.0 per cent of sodium. Soybean meal, which is being used extensively, is listed by Morrison[5] as containing just over 0.5 per cent. However, as pointed out by Ewing,[2] "It should be clearly borne in mind, in using an average figure for the mineral content of a feed, that the amount of these minerals in any particular lot of feed may differ considerably from the average."

In addition to added salt and the amounts of sodium contained in the various ingredients of a mixed feed, it is not unlikely that a great many of the mixed feed processors are using various sodium compounds as part of the mineral addition which such feed ordinarily contains.

TABLE 6—Effect of Carbonated Water

PEN	NO. OF POULTS	METHOD OF ADMINISTRATION	RESULTS
1	15	Carbonated water as drinking water	All dismissed as normal in thirty days.
2	15	Carbonated water and distilled water, equal parts as drinking water	One dead of injury in 5 days; remainder dismissed as normal in thirty days.
3	15	Tap water Control	All dismissed as normal in fourteen days.

TABLE 7—Effect of 0.75 Per Cent Solutions of Various Sodium Compounds When Used as Drinking Water

PEN	CHEMICAL USED	NO. OF POULTS	RESULTS
1	Sodium Citrate	30	Nineteen dead with typical edema and ascites in twenty-two days; 9 dead during the period of various nonspecific causes; 2 remaining killed in twenty-two days; both normal.
2	Sodium Iodide	31	Two dead in twenty-four hours, cause undetermined; by fifth day all were dead; only 2 showed edema and ascites; the remainder showed little or no macroscopic change.
3	Sodium Hydroxide 0.1 per cent solution*	31	Two dead with typical edema and ascites in thirteen days; remainder dismissed in twenty-one days.
4	Sodium Carbonate	26	Two dead in twenty-four hours, not definitely edema; the remainder drank very little and all died during the first seven days; all showed white exudate in the air sacs, and most showed white exudate over heart, lungs, or liver.
5	Sodium Sulfate	34	Eight were accidentally killed during the first forty-eight hours; in fifteen days 2 were dead, cause undetermined, and 11 were dead of typical edema and ascites; rats killed the remaining 13 at this age and autopsy of these showed 2 typical ascites cases, and 4 with hydropericardium.

*Poults in pen 3 were started on 0.75 per cent sodium hydroxide, but this, as well as 0.5 and 0.3 per cent solutions, was so unpalatable that it became necessary to resort to a 0.1 per cent solution.

Water with high mineral content is common and, as given to starting poults, may contain some sodium. We have been able to obtain but one complete chemical water analysis of a supply which was being used for a flock in which losses from this condition had occurred. This analysis showed 23.6 parts of sodium per million.

The widespread use of lye as a disinfecting agent for poultry houses and equipment offers another source of sodium. However, with the exception of that which may have been left in watering and feeding equipment, it is not readily apparent just how poults might be affected by it as it is commonly used. It appears possible that poults might obtain sufficient amounts of sodium from combined sources to produce edema and ascites.

The possibility that formaldehyde fumigation produces the condition leads also to the supposition that anything which can cause sufficient injury to interfere with the functions of the respiratory and circulatory systems might produce the disease.

CONCLUSIONS

1) Sodium chloride and sodium bicarbonate, when administered with feed or water, in proper proportions, will produce edema and ascites in starting poults.

2) It is strongly suggested that other sodium compounds, when administered to starting poults, in proper proportion to feed or water, will produce edema and ascites, provided the effective level is not so unpalatable as to prevent its consumption.

3) All sodium compounds tried in this experiment appear to be lethal for starting poults at comparatively low intake levels.

4) Sources from which poults might obtain toxic amounts of sodium are discussed.

5) It is possible that formaldehyde fumigation may cause edema and ascites in poults.

6) Investigations should be made of the sodium intake of poult flocks reporting losses from edema and ascites.

References

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[2]Ewing, W. Ray: Handbook of Poultry Nutrition. W. R. Ewing, Publisher, 1941.

[3]Witter, J. F.: A Preliminary Report on the Injurious Effects of Sodium Bicarbonate in Chicks. Poult. Sci., *15*, (1936): 256.

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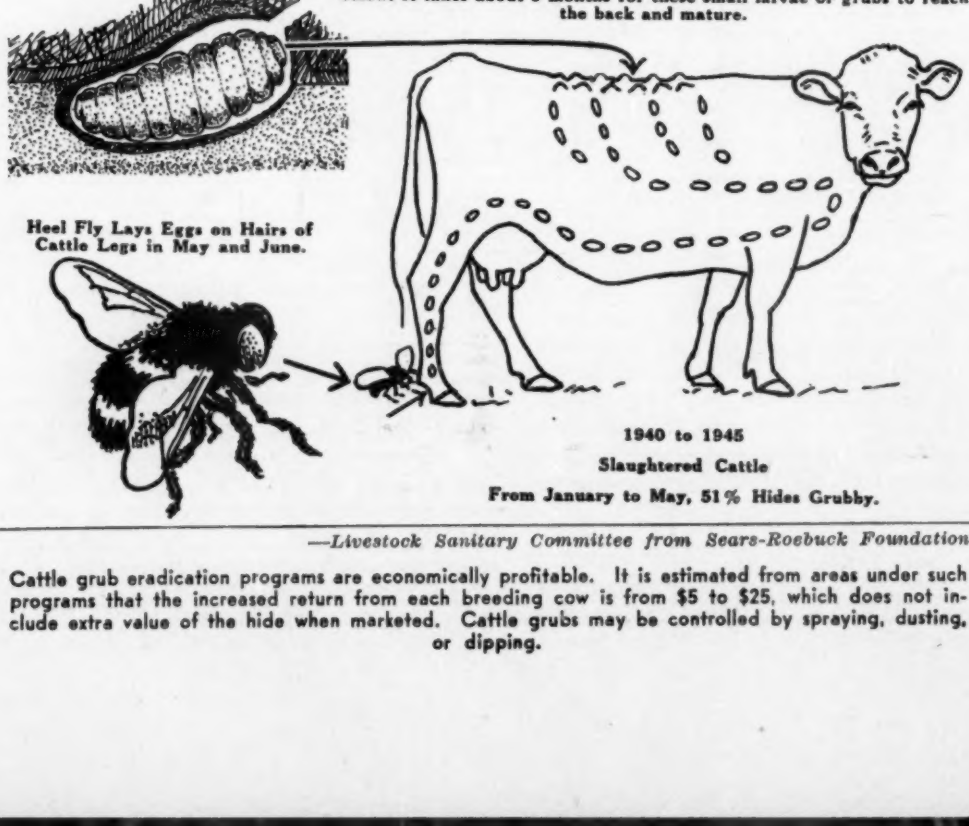
PATH OF VERY SMALL GRUBS

The eggs laid on hairs of cattle legs hatch into small larvae that enter the skin and travel through the body as indicated in the cattle outline below. It takes about 9 months for these small larvae or grubs to reach the back and mature.



—Livestock Sanitary Committee from Sears-Roebuck Foundation

Cattle grub eradication programs are economically profitable. It is estimated from areas under such programs that the increased return from each breeding cow is from $5 to $25, which does not include extra value of the hide when marketed. Cattle grubs may be controlled by spraying, dusting, or dipping.

CLINICAL DATA

Clinical Notes

Pigs raised free from parasites average 28 lb. more at 4 months than pigs raised in an old hog lot.

Until the pigs begin to eat, it costs just as much to feed a sow raising 5 pigs as a sow raising 12 pigs.

Streptothricin has been used in experiments as a treatment of tuberculosis induced in guinea pigs, but it is poorly tolerated and it has demonstrated no antituberculous qualities.

From a large group of sulfones and sulfonamides used, only one (irgafen) showed any effect on experimental tuberculosis; a slightly favorable retardation was noted when it was used by Smith and McClosky. —*Am. Rev. of Tuberc., Oct., 1945.*

Promin, a chemical distantly related to the sulfonamides, has brought improvement in 137 cases of leprosy. Although it comes closer to being a cure than anything previously known, it cannot be called a specific for the disease.—*Sci. News Letter, Oct. 27, 1945.*

Staggers in Pigs

The most common cause of stagger in pigs is vitamin deficiency, according to Dr. R. L. Mitton (*J. Dept. Agric. S. Australia*). Pigs fed exclusively on cereal grains and meat meal will quickly develop a deficiency of vitamins A and B, and, unless these elements are supplied, staggers and death are commonly seen. The best source of these vitamins is young grass, but almost any green feed will do.

A pituitary extractive, used on sheep in Russia, steps up reproduction more than 50 per cent.

One safeguard of the health of the dairy herd is to keep visitors out of feed alleys, feed rooms, and hay mows.

Trichomonas foetus has been found in phagocytes; Hammond and Bartlett report observing organisms that were still motile when examined.—*From J. Parasitol., Feb., 1945.*

Influenza vaccine of the type used on army personnel is becoming available for civilians. The influenza virus is grown on chicken embryos, and it protects against types A and B. These are the types which have caused epidemics in recent years.—*Science, Nov. 23, 1945.*

Preventive Antirabic Vaccination in Algeria

Since 1929, 50,700 dogs have been vaccinated or revaccinated against rabies before being bitten. During the first ten years (1929-1939), there were three failures nine to eleven months after vaccination. In the last five years (1939-1944), there were no failures observed out of 18,200 dogs vaccinated. During 1944, 3,718 bitten persons were treated with phenicated antirabic vaccine; there were no failures or paralytic accidents. Five of these showed sudden syncope several minutes after receiving the injection—1 was alarming—but all were able to resume their occupations after thirty minutes to twenty-four hours.—*From Rapport sur le Functionment de l'Institute Pasteur d'Algerie en 1944.*

The Incidence of Intestinal Protozoa in the Dog

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ONE OF THE FIRST requisites in determining the magnitude of a parasite problem is an accurate knowledge of the incidence of the parasites involved. The incidence of parasitic infection may not be realized even where large numbers of routine laboratory examinations are made. This may be due to the inadequacy of the procedures to disclose unsuspected parasites, parasites that occur rarely, or parasites not previously known to have existed in an area. In view of these facts, a survey was made to determine the incidence of protozoan infection among a group of dogs in this area.

For this survey the parasitic protozoa were separated into three groups according to their primary location in the host. The largest group was the intestinal protozoa, the next the protozoa of the blood, and last those that are found principally in other parts of the body. Several intestinal protozoa, said to be parasites of the dog, have not been identified at our laboratory, and some of these are reported to be pathogenic under certain conditions. There is no apparent reason why these parasites might not become established in a new locality and prove to be responsible for enteritis of unknown etiology.

Canine coccidiosis, long recognized in this area, is routinely diagnosed at our laboratory. In 1942, Koutz and Rebrassier[1] reported an incidence of 5.32 per cent infection with coccidia among 1,486 canine fecal specimens. Several other studies of the incidence of canine coccidiosis have been made in recent years. Lee's report[2] in 1934, indicated a 13.8 per cent incidence of coccidiosis among the dogs presented at Iowa Veterinary Clinic from 1920 to 1932. Gassner,[3] in 1938, found that 79.0 per cent of 320 dogs examined at Fort Collins, Colo., were infected with coccidia. Smith,[4] in 1942, determined that the incidence of canine coccidiosis did not exceed 5.0 per cent of over 200 fecal specimens examined. In these studies the parasites were of the genus Isospora, the species being *Isospora felis*, *Isospora rivolta*, and *Isospora bigemina*. *Eimeria canis* occurs only in isolated cases in the United States. The coccidia are the only protozoan parasites for which surveys of their incidence in canine hosts have been made.

Other intestinal protozoa observed in dogs within the United States and Central America include *Endamoeba histolytica*, *Giardia canis*, and *Trichomonas felis*. There have been only scattered reports of the natural occurrence of these protozoa. None of them has been identified at our laboratory previous to this survey. The pathogenicity of *E. histolytica* for the dog has been established by Faust[5] and also by Swartzwelder.[6] Hegner[7] observed that *G. canis* may become pathogenic in dogs when certain dietary conditions exist. *T. felis* has never been isolated in dogs in the United States, but it has been found in that genus in Panama, and closely related species have been observed in man and prairie dogs in the United States. The occurrence of this parasite in dogs has been associated with outbreaks of dysentery by Brumpt[8] and other European authors.

This survey has been made on the canine patients presented at the veterinary clinic, The Ohio State University. A total of 113 dogs has been examined to determine the incidence of the seven protozoa mentioned. Thirty duplicate examinations were made to gather evidence concerning the influence of successive examinations on the accuracy of laboratory diagnosis.

The diagnosis of protozoan infection is made by demonstrating the presence of the organism. The oöcyst is the form commonly found in coccidiosis. Both cystic and trophozoic forms of *G. canis* are found in the feces of infected dogs, while only trophozoites are known to occur naturally in dogs infected with *T. felis* and *E. histolytica*. Cystic forms are said by Craig to occur most often in solid stools, while trophozoites are usually seen in fresh, fluid feces. Due to this fact, both solid and fluid fecal specimens were collected for examination. Specimens were taken from normal dogs as

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well as from those showing evidence of parasitism.

A total of 81 solid and 56 fluid specimens was examined. The solid specimens were prepared for microscopic examination in the manner suggested by Faust and his coworkers.[9] Basic fecal suspensions were prepared by mixing 5 Gm. of feces with 20 cc. of physiological saline solution. This mixture was strained through a copper screen having 40 meshes to the linear inch. After 2 cc. of basic suspension was centrifuged twice in tap water, zinc sulfate solution* was added, and the material was again centrifuged for a minute at 2,500 r.p.m. Wassermann tubes were used as centrifuge glasses. Four loopfuls of the surface material was mixed on a glass slide with a drop of D'Antoni's iodine stain† and examined at a magnification of x100. Final identification was made with the 4 mm. objective and a 10 x ocular.

TABLE I—The Incidence of Infection with Coccidia

Species of Coccidia	Type of Preparation Simple Smear	$ZnSo_4$ Centrifugation	Number Dogs Examined	Infected (%)
Isospora felis	1	3	113	3.5
Isospora rivolta	0	5	113	4.4
Isospora bigamina	0	3	113	2.6
Eimeria canis	0	0	113	0
Total	1	11	113	10.6

In order to classify the oöcysts as to genus, the unsegmented ones were placed in a 2.0 per cent solution of potassium dichromate and examined after twenty-four hours. They were then identified on the basis of the number of sporocysts per oöcyst. The species of Isospora were differentiated on the basis of the size of the oöcyst.

The fluid stools were placed in an incubator, kept at 37 C. prior to preparation, and examined within two hours following passage. They were prepared for microscopic examination by smearing a fleck of feces in a drop of physiological saline solution across the surface of a glass slide. One half of the smear was stained with D'Antoni's iodine, and the stained and unstained portions of the smear were covered with separate coverslips.

The incidence of coccidial infection among the 113 dogs examined was found to be 10.6 per cent. In all but 1 of the 12 coccidial infections that were diagnosed the oöcysts were identified in centrifuged preparations. Only members of the genus Isospora were found. The results observed for the coccidia are shown in table 1.

Twenty dogs, which constituted 17.7 per cent of those examined, were found to be harboring the intestinal flagellate, *G. canis*. The motile trophozoite was found in 7 dogs in simple smear preparations while the cystic form of *G. canis* was identified in centrifuged preparations from 13 dogs. The results calculated for the incidence of giardial infection are demonstrated in table 2.

No identifiable forms of *E. histolytica*, *T. felis*, or *E. canis* were observed. It should be noted that only 56 specimens were prepared in a manner that would best demonstrate the trophozoites of *E. histolytica* and *T. felis*, and only 87 specimens were properly prepared to adequately show oöcysts of *E. canis*.

TABLE 2—The Incidence of Infection with Giardia Canis

Forms Identified	Type of Preparation Simple Smear	$ZnSo_4$ Centrifugation	Number Dogs Examined	Infected (%)
Cysts	0	13	113	11.5
Trophozoites	7	0	113	6.1
Total	7	13	113	17.7

The results of the duplicate examinations on 22 dogs illustrate how repeated examinations can increase the accuracy of infection determinations. In 8 of the dogs there were variations in the results of examinations when compared with those previously performed. There were 3 cases of *G. canis* infections that were diagnosed after at least one previous examination had been negative. In 5 cases, Giardia or coccidial cysts were identified when previous smear preparations had been negative.

In analyzing the results of this survey, it is found that no similar reports of Giardia infection are available for comparison. It is noted that all of the dogs found to be harboring *G. canis* were presented by different owners. Therefore, transfer of the organisms among these 20 dogs before

*Zinc sulfate solution is prepared by placing 331 Gm. of $ZnSO_4 \cdot 7H_2O$ (granular U.S.P.) in a graduated cylinder and adding distilled water to the liter mark. This gives a specific gravity of 1.180.

†To prepare: See Todd and Sanford's Clinical Diagnosis by Laboratory Methods. 10th edition. p. 488.

their arrival or while at the clinic was considered to be quite an impossibility. These organisms have not been detected in our routine examinations prior to this time, since the flotation and centrifugal procedures commonly used tend to destroy the motility of Giardia trophozoites. The cystic form of *G. canis* is only recognizable after association with the trophozoic form and careful study.

In 7 of the 20 cases of Giardia infection a diarrhea was present. However, in 2 of these dogs there was a concurrent coccidial infection. Hegner[7] had concluded, as a result of his experiments with flagellated protozoa, that these organisms may reproduce rapidly under certain dietary conditions and become responsible for enteritis characterized by diarrhea. The results of this survey warrant an intensive study of the ability of *G. canis* to produce disease.

The results concerning the incidence of canine coccidiosis compare favorably with nearly all similar studies made in the United States in the past ten years. It was unusual that the three species of Isospora should tend to occur at the same rate. No difference was noted during this study in the ability of the Isospora species to produce enteritis.

It has been said that the correct incidence of *E. histolytica* in any of its hosts is known only where routine examinations are made. Study of the extent of amebic infection in dogs should include the possibility of a seasonal variation in occurrence. Other writers have noted the association of canine and human amebiasis. There has been an average of 1 human case of amebiasis diagnosed annually during the past several years at University Hospital, Columbus, Ohio; however routine examinations of a large number of patients are not conducted at this hospital.

SUMMARY

In a survey of intestinal protozoa in the canine patients presented at the veterinary clinic, The Ohio State University, an incidence of 10.6 per cent coccidial infection was found to exist in 113 dogs examined. Three species of the genus Isospora were identified. Twenty dogs or 17.7 per cent of those examined were found to be infected with *Giardia canis*, a flagellated protozoa. In about one third of the Giardia infections there were symptoms of enteritis, characterized by the occurrence of diarrhea. It seems that these results should warrant future investigation regarding the pathogenic rôle of *G. canis*.

No forms of *Endamoeba histolytica*, *Eimeria canis*, or *Trichomonas felis* were found. However, not over 56 of the fecal examinations were prepared to demonstrate trophozoites of *E. histolytica* or *T. felis*, and only 87 preparations were considered to be adequate to show oöcysts of *E. canis*.

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Swine Tuberculosis

During the year which ended July 1, 1945, market hogs showed 10 per cent more tuberculosis than in the previous year. About 1 hog in every 12 slaughtered showed lesions of tuberculosis. In most cases, the lesions were limited to the head, so that very few entire carcasses were condemned. The infection is of avian origin, and it was found that half of the poultry flocks suspected as sources maintained tuberculous birds more than a year old.

Phillips has reported (*J. Dai. Sci.*, Nov., 1945) a method of coloring semen with neutral red, with Sudan III, and with Nile blue sulfate to avoid errors, especially when semen from two breeds must be carried in a single container.

Sulfapyridine in Mastitis

When sulfapyridine was fed at the rate of 5 or 6 Gm. per 100 lb. of weight (given in 3 fractions daily for four to six days) to 12 cows with a latent infection of streptococcus and staphylococcus organisms, the following results were observed by Jaquette, Kleckner, and Klein: Symptoms disappeared in all catarrhal cases, but infection persisted in two; clinical recovery was noted in 3 of 7 cases of parenchymatous but infection persisted in all; a total of 17 quarters was treated, and of these, 10 were relieved of symptoms but only 2 of infection.—*Univ. of Penn. Bull.*, **27**.

Penicillin in Blackleg

In April, on a farm where the loss of several calves had been reported one month previously, two more became seriously ill for twenty-four hours with symptoms characteristic of blackleg. One was sacrificed for postmortem examination. Tissue specimens were positive on laboratory study. I administered penicillin (100,000 units), intravenously to the other calf, which had been sick with blackleg since the morning before, but it died about 9:00 p. m. of the same day that the penicillin was administered. All of the cattle were vaccinated with blackleg bacterin.

The following morning another calf, about 4 to 5 months old, was found to be sick in the same degree as the one previously mentioned. I administred 70,000 units of penicillin, intravenously, and 30,000 units, intraperitoneally. The animal became prostrate late that afternoon, continuing in that condition throughout the next day, but on the morning of the third day, the farmer found the calf "up and cutting capers." The calf has been normal since, and there have been no more cases of blackleg.

In May, another farmer reported the loss of some calves. He reported lameness and swelling of the hind quarters with gas under the skin. On the day that he reported the losses, he presented a calf as noticeably ill as the others had been in the early stages. On the basis of history only (no laboratory report), I administered 200,000 units of penicillin intravenously. The calf lived for four days and then died with the characteristic symptoms and external appearance of the others. There was no opportunity to get tissue specimens for the laboratory. All calves were vaccinated and no other cases occurred.—*R. O. Rydell, D.V.M., Wheaton, Minnesota.*

The Liver of Hibernating Mammals

The liver of hibernating rodents and carnivores is larger, in proportion to their size, than that of other animals of the same living weight and of the same zoölogical groups, and the spigelian lobe has an accessory lobule of sufficient dimensions to be called the hibernal lobule. These increments are said to provide the hepatic capacity required to store abundant reserves during the long period of inactivity.—*Compt. rend. Acad. des sci.*, **214**, (1942): 185.

X-Ray Pictures in Diagnosis

A laudatory review of "Radiology in Canine Medicine," by George B. Schnelle, Angell Memorial Animal Hospital, in the *Veterinary Record* of Sept. 15, 1945, praises the author for having "produced a valuable guide to the small animal practitioner," just as others who have reviewed or read this fine book have done.

The British reviewer, however, goes a step farther. He seizes the occasion to point out that prints of x-ray pictures are over-touted as guides to diagnosis, emphasizing that, except in the case of gross changes or foreign bodies, only the original film amounts to much as a guide in diagnosis. Who'll disagree with that self-evident truth? Certainly not the editor who has to confess that he has pawed, selected, scanned, and paid for x-ray pictures for quite a spell in the vain hope that someone somewhere, somehow might perchance connect the landscape with the text, and certainly not the bewildered practitioner who remarked the other day that he never could tell an x-ray picture from a photostatic copy of the moon. Can you? The fault is not so much in the artistry as in the strange failure of authors to translate shadows and shadowy blends into words. Or, can that be done—like the microphotographers who do so well nowadays?

Rabies in Cattle

S. F. STAPLETON, D.V.M.
Americus, Georgia

ALL OF THE CASES of rabies in cattle that I have observed in previous years have exhibited definite viciousness and nervousness and have died in forty-eight hours or less. This year (1945), however, in Sumter county, rabies in large animals has been due, in part, to the bite of rabid foxes, which have been present in large numbers.

Last March, one of my clients had a cow that showed typical rabies (viciousness, bellowing, paralysis, and death on the second day). Several days later, he had another case that was less typical. This one showed extreme rectal tenesmus, voided little by straining, and was but slightly restless. When offered feed, the animal put her head into the stanchion and ate. I saw her three times on successive days. The last time, she still showed no signs of rabies, except slight nervousness. She was easily handled and was still eating. She died on the fifth day after the first appearance of rectal tenesmus. The Georgia Department of Health rendered a positive report of rabies, and Dr. T. F. Sellers, director of laboratories, wrote:

I appreciate the additional reports on your observations on cattle.

I take it that the Austen cow is expected to die, if not already dead. We would indeed be glad to get the brain and to send a portion of it to Dr. Johnson with your clinical remarks. I suggest that if you encounter any other cattle with rectal straining phenomena, but which do not die, you get for us samples of blood so that we can forward these to Dr. Johnson for the measurement of rabies antibodies.

I am quoting a part of Dr. Johnson's letter to me relative to your report. It will be interesting to follow up his suggestion that cattle having the rectal straining symptoms may have been those bitten around the legs rather than around the head.

"The correspondence of Dr. Stapleton of Americus, Ga., is very interesting. His description of the paralysis in cows is very much like that of the derriengue infection of cattle in Mexico. I must admit that I have never had the opportunity to see a rabid cow and the descriptions I have of the disease in this animal are neither extensive nor numerous. From our experience with dogs, I would imagine that the course of the disease is probably explained by infection from bites in the hind legs as dogs inoculated in the hind quarters do show this type of clinical picture."

s/T. F. SELLERS, Atlanta, Ga.

Dr. Harold Johnson, who is head of the rabies institute, Montgomery, Ala., may be correct, but I have wondered if the answer does not lie in the possibility that the virus, having passed from one fox to another, has become attenuated to the extent that it no longer produces typical symptoms. If this proves to be true, we shall probably have some cases of rabies that do not die. On May 20, I saw an Aberdeen-Angus cow with rectal tenesmus that fought at calves but was quiet otherwise. She died that night but no laboratory examination was made. The next week, I was called to see a cow on pasture, separated from others by a wire fence. A calf had died on this farm two weeks before, after having shown rectal straining, some viciousness, and paralysis before death. No laboratory examination was made. The cow had strained for two weeks and three days before I saw her. The straining persisted for two or three days longer, then the cow became apparently normal. When examined, she was showing typical furious rabies (bellowing, fighting, pawing) and had run the overseer from the pasture. The laboratory report was positive.

On another farm on May 10, I was shown a cow with no symptoms except rectal tenesmus. She lived five days, and the laboratory report was positive. The owner reported that a similar case two weeks before had shown no viciousness, and no abnormal conditions *post mortem*. In addition to these, I have had 6 other fatal cases of rectal straining that were not submitted for laboratory diagnoses.

Since the attention of the Albany laboratory was called to these atypical cases of rabies, subsequent inquiry has revealed that other practitioners have had the same experience.

In 1943, accidents killed more than 17,000 farm people and injured 1,500,000.

Anthrax in the United States

From a survey of the history, incidence, and distribution of age-old anthrax in this country, documented by C. D. Stein, of the Pathological Division of the U. S. Bureau of Animal Industry (*Vet. Med.*, October, 1945), one may not only gain the information promised in the title but also learn the obligation one state owes to the others in the matter of maintaining a competent live-

YEAR	S.D.	NEB.	IOWA	MINN.	TOTAL
1937	1,079	462	28	23	1,592
1938	110	20	9	13	152
1939	107	25	1	0	133
1940	128	27	3	3	161
1941	152	17	2	6	177
1942	61	3	3	3	70
1943	38	5	11	2	56

stock sanitary service. Though the early history of the disease in America is vague, the presumption is that it was implanted in the Mississippi Delta and the valley of the Rio Grande from the Old World by ships of the primitive settlers and explorers. The tenacity of anthrax, once implanted in a region, is shown by the outbreaks suffered by Louisiana livestock up to the present time. Stein's survey (*loc. cit.*) of the period, 1916-1944, reports "many outbreaks, some widespread." But the most significant fact set down is that the disease is now nation-wide, for, during the period covered, there have been visitations of anthrax in all but five states: Arizona, Maine, Indiana, Michigan, and West Virginia. The heaviest losses were suffered in Arkansas, California, Louisiana, Nebraska, South Dakota, and Texas, with South Dakota and the adjoining Nebraska counties the highest. From 1933 to 1944, widespread outbreaks involving 61 counties in South Dakota put that state at the top of the list among 405 counties in 37 states. In 1937, South Dakota showed more than 1,000 premises in 46 counties infected. In that same year, Nebraska suffered its most devastating outbreak of record in the counties bordering South Dakota. Iowa and Minnesota also were involved in the spread of the infection. The incidence in South Dakota and Nebraska, the author says, "has apparently decreased in recent years due to preseasonal vaccination." The table depicting the anthrax outbreaks in these four states shows the force of livestock sanitary medicine.

Rabies Can Be Conquered

A full-page, feature article in *Country Gentleman* under this title goes on to say: "A significant sign of the times is the increasing responsibility being placed on veterinarians. . . .To progressive veterinarians, the present reported number of up to 10,000 cases of rabies a year does not look too big to handle, compared with the bovine tuberculosis campaign in which more than 20 million tuberculin tests were applied in some years. . . . Enough scientific knowledge to do a thorough job is already at hand. When public sentiment favoring intensive rabies-control measures becomes general, rapid progress will follow."

Drying-Off Cows

Experiments by Eckles at the University of Minnesota show that it is possible to dry off cows milking up to 40 lb. simply by milking them out as usual, thoroughly sealing the end of the teat with collodion, and letting them stand. If the udder is normal, no ill effects are noted.

"If a cow is infected with mastitis, then this procedure may need to be varied somewhat. A competent veterinarian should be consulted, and his advice followed in drying up cows that have infected udders," says Prof. K. L. Turk.

Sulfa Drugs in Certain Poultry Infections*

J. C. Hammond (*Poultry Sci. 24*, (1945): 14-15) reports favorable results obtained in treating a large flock of pullets with sodium sulfathiazole given in the drinking water. In the course of the treatment, reactors to tests for *Salmonella pullorum* and/or *gallinarum* were removed from time to time.

Hawkins and Kline, of the Michigan Agricultural Experiment Station (*Ibid.* 277-281), obtained some favorable results from sulfamethazine in coccidiosis of 4-week-old chicks, treated no later than four days after exposure. The treatment interfered with normal gains, however. Induced neurolymphomatosis, by E. D. Asplin (*Ibid.* 379-382), "proved sensitive to sulfathiazole." The test is said to have supported the contention that the agent of neurolymphomatosis is involved in typical fowl paralysis.

*Abstracts from Exper. Sta. Rec. 93, (Oct. 1945): 622 and 625-626.

ANTU (Alpha-Napthyl-Thiourea) Raticide

This new product acts almost exclusively on rats, leaving human beings and most other species unharmed. It kills rats by producing edema of the lungs; the rats practically drown in their own body fluids. ANTU is a fine, gray powder with little odor or taste, which may be mixed with finely ground corn or wheat, sprayed or dusted on cut-up fruits or vegetables, dusted on the surface of water which the rats drink, or be blown into the rat holes with a dust gun. One pound of the powder can kill 300,000 rats.—(*Sci. News Letter, Oct. 6, 1945*).

Streptomycin in Tuberculosis*

The antibiotic isolated from *Actinomyces griseus*, tested for its antituberculous property by Smith & McClosky, of the U. S. Public Health Service (*Pub. Health Rep. 6*, Sept. 28, 1945 : 1129--1138), proved to be 10 times more effective than promin in guinea pigs which had been artificially infected with the human type of *Mycobacterium tuberculosis*. The treatment lasted ninety days. The dose of streptomycin was 5,000 units given intramuscularly and of promin 0.5 Gm. given orally. The dose of the former was less than 1/20 of the maximum tolerated dose and that of promin about 1/2 of the maximum dose. Larger doses, better methods of administration, and a combination of the two are among the suggestions made. Streptomycin and promin appeared to have a synergistic action.

BCG Vaccination

Though more than 2 million children and some adults have been vaccinated against tuberculosis with BCG since 1922, most of the work has been done in Europe; less in the Western Hemisphere. It has not been popular in the United States because its use has not been well controlled, and that is not easy to achieve. Until a controlled method of use has been employed, its merits cannot be definitely determined. Such experiments are under way by the Office of Indian Affairs, USDI. The work of Dr. E. A. Watson, in Canada, and of the U. S. Department of Agriculture is well known to have given unfavorable results in cattle.

*Described under Streptotrichin, *J.A.V.M.A.*, 104, (Mar. 1944) : 157.

Carbon-Dioxide Treatment for Infantile Paralysis

Drs. B. J. Aymond, R. V. Platou, and G. Peyton Kelly, Tulane University School of Medicine (*Sci. Digest, 18,* (Dec. 1945) :16), are quoted as having obtained remarkable results in the treatment of 13 patients with infantile paralysis at Charity Hospital, New Orleans, with inhalations of carbon dioxide in oxygen. The response is pronounced "impressive." In all but 1 case there was prompt relief from pain, rapid return of muscle strength, and early functional improvement. The patients volunteered the information that they were well. The experience was thought worthy of reporting even though the number of cases was small.

Cholera in Turkeys

Sanitation and hygiene play an all-important rôle in preventing fowl cholera. The University of Illinois recommends that turkey owners secure the services of a qualified veterinarian to determine the cause of trouble. Turkeys should be segregated from all other fowl, held under strict sanitary precautions, allowed plenty of range which can be rotated at two-week intervals, and droopy or listless birds should be removed from the flock promptly.

Brucella Vaccination

By comparing 35 vaccinates and 23 controls, Birch, Gilman, and Stone found that approximately half of the vaccinates eventually acquired the infection and became spreaders but did not, as a group, show extensive outward manifestations. They conclude that vaccination is much more effective in delaying the development of brucellosis and in softening its effects than in its actual prevention.—*From Cornell Vet., April, 1945.*

(This is discussed also in the report of the Committee on Brucellosis in the November, 1945, JOURNAL.)

SURGERY & OBSTETRICS

AND PROBLEMS OF BREEDING

Abortion in Sheep Following the Administration of Phenothiazine

B. L. WARWICK, D.V.M., Ph.D., R. D. TURK, D.V.M., M.S., and R. O. BERRY, Ph.D.
College Station, Texas

PHENOTHIAZINE, which has been satisfactorily used as an anthelmintic with very large numbers of animals, particularly sheep and goats, is generally considered to be nontoxic in the dosages usually recommended. It also is generally accepted that phenothiazine may be safely administered to pregnant animals. For example, in Stewart's excellent review of the literature on the use of phenothiazine in veterinary practice,[1] the following statement is made: "Probably the greatest advantage of phenothiazine over other known anthelmintics is the apparent safety with which it may be administered to pregnant animals, even those late in pregnancy . . ."

We have used phenothiazine repeatedly in our genetic experimental flock of sheep and goats, routine treatment being given at more or less regular intervals, with little regard as to whether pregnant animals were included. We have not always recorded the dates on which the animals were drenched, or the individual identity of the animals drenched on a particular day, and so we do not know, in many cases, the exact stages of pregnancy of the treated animals. Therefore, an occasional stillborn lamb could have been the result of phenothiazine without exciting suspicion. However, no evidences of toxicity had been noted prior to the treatment administered to sheep on Dec. 14, 1944. On this date, although lambing had started, all the pregnant ewes were treated except 13 ewes which were confined in individual lambing pens and 22 ewes which had been selected for resistance to parasitism.

Each of 154 pregnant ewes received approximately 25 Gm. of phenothiazine as a drench. Bentonite was the agent used for putting the phenothiazine into suspension in water. Twenty-three of these ewes were from the 138th day to the 145th day after breeding, which is within the last ten days of the usual gestation period. As shown in table 1, 12 of these 23 ewes gave birth to either premature or stillborn lambs within four days after being drenched with phenothiazine. Three of the premature lambs were not found as they were lost in the pasture. Six of the 12 ewes suffered dystocia of varying degrees of severity, 2 of which required embryotomy. Of the 131 ewes, at fifty-two to 137 days after breeding, all but 6 lambed normally. Of these 6 cases of abnormal parturition, each ewe carried the fetus or fetuses to term, which was from twenty-seven to eighty-seven days after treatment. There was no evidence of prenatal death and degeneration of the fetuses. If phenothiazine treatment had been responsible for any of these 6 cases, we would have expected some evidence of earlier death and degeneration. In the absence of such evidence, we are inclined to the view that these were sporadic cases not caused by phenothiazine.

Of the 13 ewes confined to individual lambing pens and not treated December 14, 2 were at the 148th day, 4 at the 146th day, and 7 at the 145th day, respectively, after breeding. All lambed normally. The 22 ewes which were not treated because of the parasite resistance experiment were from the 98th to the 136th day after breeding. One dystocia case with a stillborn pre-

Animal geneticist, Agricultural Experiment Station, (Warwick); head, Department of Veterinary Parasitology, School of Veterinary Medicine (Turk); and associate geneticist, Agricultural Experiment Station, (Berry); A. and M. College of Texas, College Station.

mature lamb at the 138th day occurred in this group. The remaining 21 ewes lambed normally.

It is evident from the experiences reported herewith that phenothiazine may be toxic when administered to ewes during the later stages of pregnancy. Swales and Collier[2] reported dystocia with stillborn twin lambs the day after treatment of their ewe 4 with 60 Gm. of a drug mixture containing 80 per cent phenothiazine. These authors state: "It is possible that the laxative effect of the treatment may have accelerated the oncome of parturition in the case of ewe 4, and may have been a contributing factor in the loss of the two foetuses." Boughton states that he has observed abortion in 2 range flocks of sheep following the administration of phenothiazine drench during the lambing period. In both instances, stillborn and apparently premature lambs were found within four days following treatment. Both flocks were pasture bred, and no detailed breeding records were obtained.[3] Habermann, Foster, and Hummon[4] gave phenothiazine to ewes as close as two days before normal parturition. The dosage they used was 25 Gm. per animal, given in a mixture of molasses and feed. They also used phenothiazine without molasses, with no apparent harm. Included in their report was 1 ewe which received 25 Gm. of phenothiazine by capsules two days before giving birth to a healthy lamb. They also gave 15 Gm. per day in molasses-grain feed to 1 sheep for twenty-one days immediately preceding normal parturition. Evidently, phenothiazine is not always toxic to animals late in pregnancy. It may be true that it is usually harmless, and proves toxic only under certain conditions. If this assumption is true, we still are ignorant as to what conditions predispose the animals to the toxic effects of phenothiazine. It is certain, however, that there is a period late in pregnancy when this drug *may* cause abnormal parturition. Since the maximum normal gestation period of sheep is apparently about 158 days, the period of danger is approximately two to three weeks prior to parturition. Figured from date of breeding, this period extends from about the 137th day to the end of the gestation period.

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TABLE 1—Lambing Records of Ewes Treated, December 14, 1944*

No. of Ewes	Days Between Breeding and Date of Treatment	Parturition		
		Date (Dec.)	Days after Breeding	Notes
2	145	16	147	Stillborn, 1 dystocia.
2	145	17	148	Stillborn, 2 dystocias (1 embryotomy).
1	144	16	146	Stillborn, dystocia.
1	144	17	147	Stillborn, dystocia.
1	144	18	148	Stillborn.
1	143	24	153	Normal.
1	142	17	145	Stillborn, dystocia (embryotomy).
1	142	19	147	Normal.
1	141	18	145	Premature, lamb not found.
1	141	23	150	Normal.
1	140	22	148	Normal.
1	139	25	150	Normal.
1	139	27	152	Normal.
1	138	15	139	Premature, lamb not found.
1	138	17	141	Premature, lamb died at birth.
1	138	18	142	Premature, lamb not found.
1	138	24	148	Normal.
3	138	25	149	Normal.
1	138	30	154	Normal.
2	137	26	149	Normal.
4	137	27	150	Normal.
1	137	28	151	Normal.
1	137	29	152	Normal.
1	137	30	153	Normal.
1	136	24	146	Normal.
2	136	27	149	Normal.
1	136	29	151	Normal.
1	136	30	152	Normal.
1	136	31	153	Normal.

*In addition to the ewes included in the table, there were 116 ewes at fifty-two to 135 days after breeding. All but six lambed normally. See text for details.

Skin Grafting in a Cat

FRED KEEFE, V.S., B.V.Sc.
Boston, Massachusetts

DURING THE PAST few years, some of the most tedious and unsatisfactory cases presented have been those suffering from extensive loss of skin on various parts of the body. While such cases may recover after a long period, slow healing detracts considerably from the satisfaction derived from the recovery, to say nothing of the impatience of the clients.

Skin grafting is an old practice, but until recent years little interest was shown in its possibilities. World War II has done much to revive experiments in the various types of grafting. The Cocoanut Grove fire showed that much could be done for burns that in the past had been fatal.

An accident case was presented at the clinic of the Angell Memorial Animal Hospital immediately after being struck by an automobile. The patient, a 3-year-old tricolored cat, in fair condition, was suffering from shock and loss of blood. The skin of the left hind leg, from just above the hock to the lower third of the metatarsals, had been peeled off and left hanging. A very doubtful prognosis was given, but the owner insisted that everything possible be done to bring about recovery.

The patient was immediately anesthetized; the area involved was prepared and debrided as thoroughly as possible, and the skin flap was sewed into place with numerous, small, closely-placed, catgut sutures.

At the end of six days, the flap had become necrotic; it sloughed off, and left an extensive area of granulating tissue completely encircling the limb from just below the hock to the foot.

For the next three months, the usual methods were employed in an attempt to stimulate the growth of new skin. At the end of this period, there was still a completely denuded area, 2 inches in length, encircling the leg from just above the foot to 1 inch below the hock. At this point, we decided to attempt a skin graft,[1] proposing to improvise as we went along.

Treatment of Lateral Side.—The lateral side was treated first. An area was selected and prepared on the lateral surface of the thigh, a small nick was made through the skin, and a large area of skin about 2 inches wide and 5 inches long was separated from the underlying tissues by blunt dissection. Then commencing at the nick, a rectangular

Fig. 1—Lateral view, showing extensive denuded area between hock and foot: flap of skin lifted and folded back to show rectangular area left.

flap of skin 3/4 of an inch by 4 inches was resected, attachment being maintained at the distal end, as shown in figure 1. Laying back the flap left a rectangular denuded area on the upper thigh, which was closed by interrupted sutures (fig. 2), the procedure being facilitated by the previous extensive blunt dissection. The next step,[2] also shown in figure 2, was to form a tube of

Contribution from Angell Memorial Animal Hospital, Boston, Mass.

a portion of the flap, commencing at the attached border, the tube being of sufficient length to bridge the distance from the aforesaid attached end to the anterior edge of the denuded area.

Figure 3 demonstrates how the[3] open end of the flap was brought into apposition by a gradual downward rotation of the tubed portion, and how the flap was sewed into position, using fine sutures gently tied into place to prevent them from tearing out of the underlying granulating tissue. At this point, great care was taken to insure that the tubed portion was curved down and turned on its long axis in such a gradual manner as to preclude any stoppage of the blood supply to the flap. The leg was then dressed in the half-flexed position, using a triple layer of gauze directly over the area, covering it with a light layer of cotton, and bandaging snugly. This dressing was kept constantly moist with saline solution, and the patient placed on a high protein and vitamin diet. At the end of three days, the dressing was carefully removed, and underneath a coating of dark mucus the tube was

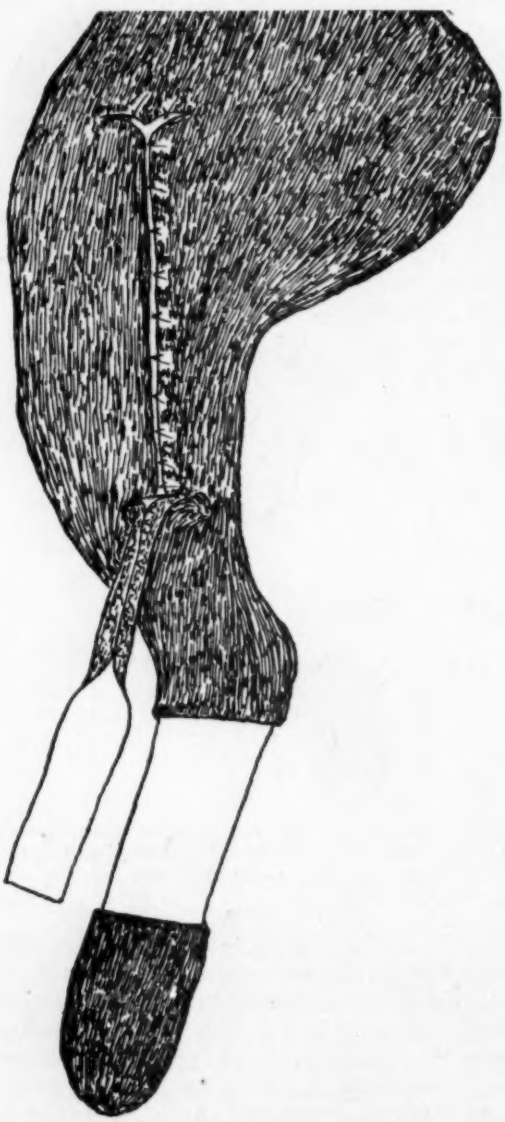

Fig. 2—Rectangular denuded area closed by fine sutures; portion of flap tubed.

Fig. 3—Tubed portion of flap curved around and down, and flap sutured into position.

found to be warm and of healthy appearance. The adjacent portion of the flap was firmly attached, swollen, and felt soft and pulpy. The lower half, however, was beginning to slough. The area was then cleaned up, smeared liberally with cod liver oil ointment, redressed, and left for four more days. On removing this dressing, the lower half of the flap had disappeared; all granulating areas were swollen and inflamed, and there were necrotic areas. This area was again thorough cleaned, dusted freely with sulfathiazole powder, redressed, and left strictly alone for one week.

This time the results were encouraging. The remaining section of the flap was healthy, firmly attached, and starting to sprout hair. The appearance was rather odd. The hair pointed upward. Small fingers of new skin were creeping up from the distal border of the denuded area and down from the distal edge of the flap. At the same time, the anterior and posterior borders of the flap were rapidly widening. Sulfathiazole dressings were repeated every five days for the next two weeks, at the end of which time the whole lateral surface of the denuded area was bridged from top to bottom.

The final operation on the lateral side was

Fig. 4—Tubed portion removed (dotted area) and small openings left closed by small sutures.

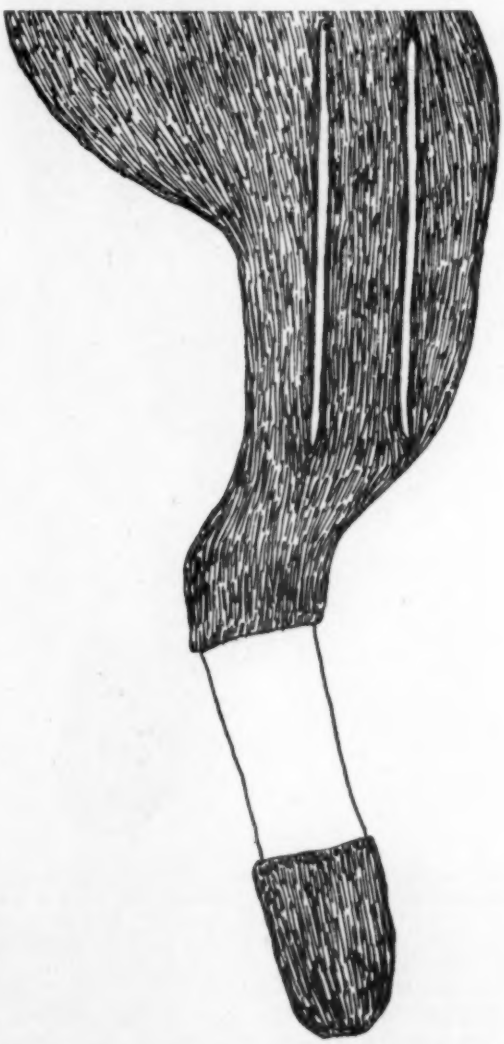

Fig. 5—Medial view: two parallel incisions made in skin; strip loosened from underlying tissues by previous blunt dissection.

the removal of the tubed portion (fig. 4). When the tube was severed at its proximal and distal attachments, the amount of hemorrhage was surprising. Small sutures were inserted to draw the open areas together, with complete healing at these two points within a week. The area on the upper thigh from which the flap originated had by this time healed by first intention.

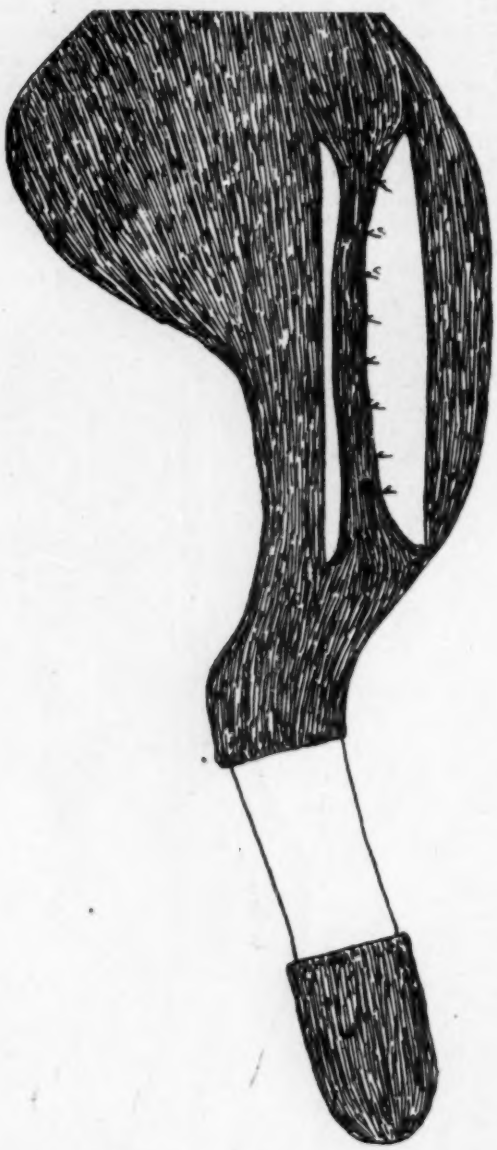

Fig. 6—Flap of skin rolled to form a tube and edges approximated by sutures. Underlying rectangular denuded area shown.

Treatment of Medial Side.— We next turned our attention to the medial aspect of the leg. This time it was decided to add another step to the procedure. Instead of producing a flap, tubing, and suturing the flap in place in one operation, the following method was used:

Fig. 7—Underlying rectangular denuded area shown closed by interrupted sutures. This completed first operation on medial side.

As on the lateral side, a large area of skin was separated from the underlying tissue on the medial surface of the thigh, care being taken to dodge the internal saphenous and femoral veins. A 1-inch by 5-inch strip was cut in the skin, but this

time both proximal and distal ends were left attached (fig. 5). The next step (fig. 6) was to roll this flap into a tube, using fine interrupted sutures to bring the edges of the flap into apposition. The underlying area, free of skin, was brought into apposition (fig. 7) as on the lateral side, and the whole area was dressed and kept moist with saline for three days. From then on, sulfathiazole dressings were applied as before. At the end of fifteen days, the tube was healthy, had grown some hair, and caused the patient no inconvenience.

The next operation (fig. 8) consisted of severing the tube at its proximal attachment, curving it down and around over the denuded area, slitting the tube open along its line of suture for a distance sufficient to form a flap large enough to extend from top to bottom of the denuded area, and suturing in place. At this point, it was discovered that due to shrinkage of the tube the flap was less than 1/2 inch in

Fig. 8—Second operation. Proximal end of tube severed; lower portion of tube slit and opened to form flap: flap sutured into place.

Fig. 9—Third operation. Tubed portion removed (dotted area) and small openings left closed by fine sutures.

width. This time the whole flap became firmly attached except for a narrow strip of necrosis along its distal border.

Fourteen days later, the tube portion was removed (fig. 9) and the lower limb dressed every five days. Two weeks later, the medial flap had united the top and bottom edges of the denuded area, and had progressed from side to side to meet the lateral flap except at two points. This left, on the antero-medial aspect of the leg, two denuded, nonhealing areas each about the size of a quarter.

The final operation consisted of taking small pin-point grafts from the skin of the abdomen, and peppering the two areas with them. Dry sterile dressings were applied, and at the end of five days, the results were amazing. The grafts, each originally about 1/8 inch in diameter, had doubled their size; those in the center had coalesced, and some near the borders had joined the surrounding skin. At the end of ten days, both areas were completely covered with skin, with small islands of hair beginning to show up where the original point grafts were laid down.

The technique used in this type of grafting is comparatively simple. A long hypodermic needle is used to pick up the skin, the point elevated, and a sharp scalpel is used to pare off a flake of skin. This is transferred on the point of the needle to the denuded area, gently patted into place with the flat side of the scalpel, and the process repeated. One interesting feature is that a small pool of serum immediately surrounds the graft when placed in its new position. The serum disappears in about fifteen minutes, and the graft begins to adhere in that short time.

Thus, after a little more than two months, the denuded area was completely covered with skin, patches of hair sprouting in all directions, and the patient was using the leg normally.

The main conclusion drawn was that the pin-point grafts were much simpler and gave quicker results than the tubed flap method. It was decided to further investigate the possibilities of pin-point grafting in small animals.

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A Precocious Heifer: Cesarean Section

Examination of an unusually precocious heifer, 13 months old, in labor six hours, having revealed the impossibility of a normal delivery, I suggested cesarean section.

After preparing the site and administering an epidural anesthetic, I made an incision from 12 to 18 inches long through the skin, musculature, fat, and peritoneum, in the usual manner, and packed sterile cotton around the opening to keep the intestine back and to absorb fluids. The search for the gravid horn revealed the case to be a left horn pregnancy. The horn was maneuvered to the incision with the posterior extremity of the fetus outward. At this point, it was necessary to enlarge the incision to permit exposure of the uterus and to place packing that would prevent the escape of fetal fluid into the peritoneal cavity. After incising the uterus, a live fetus was lifted out by the hind legs. The uterus was sutured tightly with a row of chromic catgut which was buried with a second row. The peritoneum was closed tightly to avoid seepage, and the various layers were closed with interrupted sutures of umbilical tape.

I injected pituitrin into the jugular vein to bring about a rapid constriction of the uterus and to aid the expulsion of the placental membranes; adrenalin was used to prevent shock. Liberal amounts of sulfathiazole also were placed in the uterus, peritoneal cavity, and upon every layer of the incision, which I am sure was the factor that prevented any discharge from the incision. Also, 100,000 units of penicillin were administered for four consecutive days. On the twelfth day the heifer was eating well, was active, and was well on the way to recovery.—*E. C. Lunn, V.S., B.V.Sc., Malta, Ill.*

The cost of producing milk on dairy farms in New York state has almost doubled since the outbreak of World War II, says L. C. Cunningham, of Cornell, and has now reached $3.91 a hundred pounds in the east central section of the state.

NUTRITION

Nutrition Notes

Profit can not be starved out of livestock.

Most dairy cows suffer more from a lack of adequate proteins than from lack of minerals.

During the past 25 years, the rate of feeding protein concentrates in farm rations has almost doubled.

Good feeding will begin to show results in a few days. Good breeding produces results, too, but it requires years with most classes of livestock.

The brood sow should have at least 1/2 lb. of a protein supplement each day, in addition to home-grown grains and 10 per cent of alfalfa or clover hay.

For every 100 lb. of growth a heifer makes after she calves, she is sacrificing the potential production of about 1,500 lb. of milk.

According to random reports, feeding hogs the kitchen waste of military camps during the war was held responsible for many outbreaks of hog cholera.

Cows producing a heavy flow of milk can not get enough salt from licking a block. They need the time for chewing cud.

The state of nutrition and the level of health are important in determining the severity of parasitic infection. Nutrition is also related to disease resistance — the *gamma* globulins (antibodies) are derived or elaborated from the proteins of the diet. However, many factors in addition to vigor and state of nutrition are important also.

Vitamin A Deficiency (?)

Since soybean hay has become more common as a forage for cattle and sheep, my attention has been called to the illness and ultimate death of many animals which have been fed soybean hay almost exclusively as the roughage. The illness was characterized in whole or in part by the following symptoms: normal temperature, dullness, weakness, slow weaving walk, sometimes diarrhea, anorexia, and death.

The circumstances on one farm appeared to be almost a "controlled experiment." Cows and heifers in the stanchions received the following feed: ground oats, corn fodder, silage, green alfalfa hay, and bleached soybean hay.

Across the feed alley were about 15 steers and heifers, 1 year old, more or less. They were fed soybean hay, corn fodder, silage, and ground oats. One at a time, over a period of about six weeks, 4 yearlings took sick and died. I was called when the second animal became sick. She was treated with glucose, calcium gluconate, and intestinal astringents, and the treatment seemed to postpone death for a day. I suggested that the farmer discontinue the soybean hay and feed only the alfalfa to the feeders. This he failed to do until 4 had died. The death loss stopped when alfalfa was added as roughage to the diet of the feeders. The cows across the alley had been receiving the two hays and no illness occurred in that group.

I am not certain what the reason was for the mortality, but I will venture the opinion that the cause may have been vitamin A deficiency.—*R. O. Rydell, D.V.M., Wheaton, Minnesota.*

One hundred thousand pounds is a lot of milk, but almost 1,100 Holstein-Friesian cows have produced more than that amount. Assuming that this is done in seven milking years, it means that each of these cows furnished 1 quart of milk daily for seven years for each of 20 children. The dairy cow is truly the foster mother of the human race.

Bloat in Ruminants

Bloat is not simply a matter of excess gas production in cows, because cows are able to eructate more gas than is ever formed. There appears, instead, to be interference with the eructating mechanism. This may be complete or partial choke, ruminal atony, frothy ingesta, or defective eructating mechanism.

Choke is simply a mechanical obstruction; relief of the choke will usually result in relief from the bloat as well. Ruminal atony is associated with overloading of the paunch, and possibly, also, with the production of histamine which has a depressing action on the musculature of this organ. Frothy ingesta become a factor in that there is no free gas to eructate, but the reason for the froth remains obscure. It has been suggested that saponins affect the surface tension of the bubbles, but the feeding of saponins has not been conducive to bloat. Defective mechanism may be a permanent and an organic condition, but is more likely to be transient and caused by the presence of hydrogen sulfide and/or other toxic gases.

Methods of handling cows to prevent bloat are numerous, but in the main are ineffective. A good feed of hay before putting the cows out has been recommended; it works sometimes but is not dependable unless a good quality of hay is available for more than two hours. Alternating pasture often works well; that is, putting the cows on Sudan grass through the night and on legume pasture during the day. Another variation of this plan which has been attempted, is planting grass and legumes together as pasture, but it has not been satisfactory because the cows eat the grasses first, and then fill up on the legumes when the grasses are too short. The same objection holds for pastures adjoining fields of grass and legume, under a free-choice plan. Pasturing legumes only after they have reached the blossom stage has been fairly satisfactory, but continuous day and night pasturing on younger legumes has not been, popular as the practice is. Grain feeding before pasturing has not worked well, because a considerable number of cows will eat more legume pasture after a feed of grain than they would had they not been fed the grain. It is a good plan to avoid pasturing legumes when they are wet with dew or rain, and especially when wet with frost; but the feeding of minerals to avoid bloat is classed as mere exploitation of the breeder. Bloat can be avoided by feeding the legumes as soiling crops, but this is much more expensive than pasturing.

Treatment of bloat is generally satisfactory if the veterinarian will pass the stomach tube to permit the escape of the accumulated gases. Recurrence can be avoided by introducing formaldehyde to stop fermentation. Other antiferments, less drastic, may be less irritating to the wall of the rumen. The usual home treatments are to place a wooden bit in the mouth, or to raise the front feet to promote eructation. Both work in the less severe cases but fail in emergencies. The use of the trocar should be a last resort, especially in the hands of the layman, because of the danger of subcutaneous abscesses which form when food particles escape from the rumen, but are not forced through the skin incision as well.

Many theories of bloat and an extensive bibliography for additional study are furnished by Drs. H. H. Cole, C. F. Huffman, Max Kleiber, T. M. Olson, and A. F. Schalk in the *Journal of Animal Science* for August, 1945.

Carotene and Vitamin A

The vitamin A content of blood plasma of calves up to 4 months of age varies from 7.2 to 14.0μg. per 100 cc. This is considered to be in the deficient range, when compared with calves 1 year old. Moore and Berry (*J. Dai. Sci.*, Nov., 1945) report that field-cured hay retains only 5 to 10 per cent of its original carotene content. They conclude that some vitamin A supplementation is indicated where difficulty in raising calves is encountered.

Plants are the link between minerals and livestock, says R. H. Lush (*Successful Farming*, Dec., 1945). Feed the soil for better animals; if the soil is poor, the feeds produced are of low quality and the animals suffer.

Failure to feed liberally, and failure to feed a balanced ration are the most serious faults in average feeding programs.

EDITORIAL

Pullorum Disease: Historical Sketch

Having left the control of pullorum disease largely to others, American veterinary practitioners are apt to forget that here is one of the most momentous diseases of domestic animals. Nor is it generally known among us that the poultrymen of the United States are blamed for scattering the infection all over the world through the exportation of eggs and chickens, following World War I (Barger and Card, 1943; Lesbouyries, 1945). The inescapable truth is that, at this hour, pullorum disease is the most widely spread of the avian infections and the most murderous. The raising of chickens on a large scale is impossible without first mastering that deadly plague of the newborn. That is the picture *Salmonella pullorum* has painted wherever aviculture has furnished the medium.

Pullorum disease was first described as a given disease by Rettger, of Germany, in 1889. About ten years later, he isolated the specific agent which, in 1907, he named *Bacterium pullorum* (after *pullus*, Latin for pullet, or chick). In 1909, Rettger traced the source of the infection to the ovaries of recovered fowl. For durable periods thereafter, the names for the disease were, respectively, *Rettger's disease, bacillary white diarrhea* (BWD for short) and, finally, *pullorum disease*. Under the name of bacillary white diarrhea, American pathologists (Ward and Gallagher, 1917) laid down the principles of control now universally employed—the removal of infected breeding hens by sero-agglutination. Japanese were writing fluently on the disease in 1919. In his brochure, Principal Diseases of the Poultry Yard, Moussu (1920) mentioned the heavy losses suffered from white diarrhea in the United States and points also to its prevalence in France.

In 1926, after five years of intensive study, Hinshaw and coworkers reported that 75 per cent of the poultry flocks of Kansas were affected and that 25 per cent of the hens of that state were carriers. In the early 1920's, Doyle and others in Great Britain declared that the raising of chickens, in the absence of rigorous preventive measures, was doomed. Beginning in 1917, Hadley, and, later, Van Es, Martin, Gwatkin, Brandly, Bunyea, Beaudette, Beach, Van Roekel, Jungherr, and others of this period filled many pages on its pathological anatomy, epizoötiology, and preventive technique. Meanwhile, in North and South Africa, Roumania, England, Italy, and other countries abroad, avian pathologists wrote emphatically on the terrible poultry plague American poultrymen had implanted in their respective domains through importations of eggs and chickens following World War I. Sanely viewed in retrospect, the indictment is not valid. Pullorum disease flares in noticeable degrees only where aviculture agglomerates enough chicks to provide the fuel. The disease became grave in the United States with the coming of giant incubators and prodigious expansion of poultry production, and it began to cause consternation abroad, with a similar upswing, following that war for comparable reasons. The work of Rettger in Germany and poultry pathologists in Britain, France, Holland, and Belgium shows that the specific agent was not a postwar immigrant. It was there all the time waiting for chickens to be raised on a scale above the level of the small farm flock. In view of the high incidence of pullorum disease developed in the United States in the growing poultry industry, there can be no doubt that *S. pullorum* was shipped to Europe in eggs and chickens, but the contention that the aboriginal flora would have been kinder than the American variety to the accelerated aviculture overseas is placing a lot of confidence in that famous killer of newborn chicks. Wherever there are chicks enough to stage the scene, Rettger's agent has never failed to star as the villain. We believe this is a chapter of international veterinary history that ought to be revised.

Poultry Medicine

The present period is the starting point of clinical avian medicine as distinguished from avian pathology. The former is new, the latter old. The frank, veterinary-medical writer has to admit that animals become patients only when their market value can underwrite the cost of treatment; others, unconscious of living in a world of practical economics, treat *barter* and *science* as incompatibles. Veterinarians of the past have not shirked avian medicine without good reason. It was the owners who shirked, also for good reason. The poultry flock of the farm has been a source of pin money, not of income for the farm as a whole. So, chickens were not patients until the increased demand for poultry and eggs made aviculture a profitable branch of farming and, thereby, enlarged the field of veterinary practice, as did swine and sheep and goats and fur-bearing animals in their turn.

Happily, when aviculture was transforming from *pin money* to *income* status, the avian pathologists were ready, despite the fact that theirs, in the long past, was a tolerated rather than an encouraged branch of study in the college curriculum. The material of countless bulletins, journal articles, and small manuals had to be bound between covers to put into the hands of the practitioners, when poultry medicine came rushing in. Recent examples are *Poultry Practice* in this country, and *La Pathologie des Oiseaux* (*Avian Pathology*) in France. The avian species has provided primitive insight to embryology, general pathology, genetics, bacteriology, immunology, and others, as seen in the works of Fabricus Hieronymus on embryology (1653), of Spallanzini on gastric digestion (1768), of Darwin on heredity (1836), of Pasteur on fowl cholera (1868), of Eijkman on beriberi (1897), of Ross on malaria (1898); these are among the many early studies which led to the romantic development of avian medicine. As a consequence, after years of research in the poultry yard, the avian pathologist is now able to provide the knowledge the practitioner needs in building up the new field.

When, in the teen decade, swine practice came riding in on cholera vaccination, it took thirty years before swine pathology was clarified as well as avian pathology is clarified today. From here on, avian medicine has that much advantage, thanks to the avian pathologists. But what's uppermost in mind here is that owners of animals are practical men. They did not habitually seek professional consultation for their sick chickens because the salvage did not warrant the cost, even in the case of grave epizoötics too long neglected.

This writer once had the experience of arriving at a brooder house just as the farmer had finished picking up a bucketful of dead chicks. Too late! But that was not unusual in the early days of the big hatcheries. Successful avian medicine is a matter of continuous coöperation between owner, hatcheryman, and veterinarian. It will be unfortunate if the latter does not participate.

Ancient Veterinary Medicine

Two articles translated from Sanskrit by Veterinary Officer A. Krishnaswamy, of the Madras Medical College, and published in recent issues of the *Indian Veterinary Journal* signify that the ancient mind, in respect to man's dependence upon domestic animals, was farseeing. These translations indicate that the ancient people, who developed tremendous wealth and power before western civiliation was born, showed practical knowledge and public interest in veterinary medicine. In the present chase for human welfare through international treaties, every detail of the world's work, except man's chief source of subsistence, is debated. The lack of adequate attention to veterinary medicine is worldwide.

The change in the socio-economic life of the Middle East responsible for the decline (judged by the Occidental arbiter), though never written understandingly into world history, would be a precious anthology for the modern veterinarian in his pleas for an unimpeachable agendum for veterinary science. The neglect to build and maintain a health-of-animals program of the highest possible genre appears to reach to the four corners of this self-centered world. Incredible as it may seem, the use of an inexpensive means to a fundamental end remains incomprehensible. The joke's on whom?

CURRENT LITERATURE

ABSTRACTS

Sheep Diseases

"The economical production of livestock for market depends mainly upon breeding, environment, and nutrition. The prevention of diseases, although superficially an entity, is intimately associated with these three factors. This publication will discuss sheep diseases, their various causes, and methods of control, particularly as related to herd management. Hygienic principles will be emphasized rather than specific curative agents, preventive vaccines, or bacterins, except those whose value has been clearly established." These remarks introduce a bulletin on this subject, which then proceeds to consider the whole gamut of sheep ailments. The introduction says, "This publication is not intended to supplant the local veterinarian. His assistance is recommended; often it will result in early diagnosis and, therefore, in more rapid control measures." However, the treatment of individual sheep is seldom the work of the veterinarian in the range areas, so that the discussion of most of the diseases is on the basis of flock care by the shepherd.—[*H. S. Cameron: Sheep Diseases. California Agricultural Extension Service, Circular 130, July, 1945; 36 pages. The College of Agriculture, University of California, Berkeley, Calif.*]

Sterility in Bulls

The anterior pituitary gland is the seat of secretion of those hormones which stimulate function of the testes as well as of the ovaries. FSH (follicle stimulating hormone) aids in the development of the cells in the seminiferous tubules and in maturation of the spermatozoa. LH (luteinizing hormone) is called ICSH (interstitial cell stimulating hormone). These cells secrete androgen or testosterone, which causes sex urge, the development of secondary sex characters, secretion of fluid of the secondary sex glands, and regulation of the blood supply to the testes and scrotum.

The causes of sterility may be: (1) abnormal secretion of FSH. It may be due to faults in feeding or management, such as underfeeding, overfeeding, deficiency of vitamin A or carotene, or old age. Treatment may not be advisable because of the expense of making daily injections of FSH for three or four weeks, and because successful treatment may only perpetuate the trouble. (2) Abnormal testes. (3) Abnormal secretion of ICSH. When low, it results in reduced secretion of androgen, and the animal may have semen and sperm but no sex urge. Treatment may be with any one of the various forms of LH, which stimulates the interstitial cells to produce androgen and this, in turn, increases the sex urge and the normal secondary sex responses. If sperm production is normal, androgen direct may correct the trouble; but too much of this interferes with FSH production and sperm formation. (4) Undescended testicles. Descent may sometimes be assisted by ICSH or testosterone. (5) Age reduces sex urge, and this may be helped by injecting androgen or feeding thyroprotein. These do not correct the trouble, but may keep the sire going a while longer.—[*C. W. Turner: Sterility in Bulls. Guernsey Breed. J., Oct. 15, 1945.*]

Animal Parasite List

Dr. G. Dikmans has prepared a list of animal parasites, in response to the recommendations of the Committee on Parasitology in 1943, "that the Bureau of Animal Industry of the United States Department of Agriculture be requested to publish either a bulletin or a series of bulletins containing the following information: (1) check list of parasites of domestic animals occurring in the United States indicating states in which each occurs, (2) indications by states as to which parasites are most prevalent and which are most serious, (3) species of animals which act as hosts, (4) locations of parasites in hosts, (5) intermediate hosts, if any, (6) keys for identification, and (7) descriptions." The Bureau has been unable to supply all of the information, but has agreed to a publication which complies with parts 1, 3, 4, and 5 of the Committee's recommendations.

Dr. Dikmans compiled this list in the dual capacity of parasitologist in the Zoölogical Division, Bureau of Animal Industry, Agricultural Research Administration USDA, and as chairman of the Committee on Parasitology of the AVMA. The report contains three pages of discussion, 26 pages of tables, and two pages of

bibliography. Each table consists of five columns: group and common names, scientific names, location in host, intermediate host, and geographical distribution.

There are twelve tables, and they are arranged in pairs so that odd numbered ones deal with internal parasites, even numbered with external. Tables 1 and 2 are for horses, mules, and asses, 3 and 4, cattle; 5 and 6, swine; 7 and 8, sheep and goats; 9 and 10, dogs and cats; 11 and 12, birds.

The list of parasites is complete, and it should be valuable in helping practitioners to recognize parasites, even though the identifying points are not enumerated.

This report, reprinted as a 32-page booklet, is available from the office of the AVMA, 600 South Michigan Ave., Chicago 5, Ill., at 15 cents per single copy. Ten or more copies, 10 cents each.—[*G. Dikmans: Check List of the Internal and External Animal Parasites of Domestic Animals in North America. Am. J. Vet. Res., 6, (Oct., 1945): 211-241.*]

BOOKS AND REPORTS

Veterinary Clinical Pathology

In writing this introduction to veterinary clinical pathology, the author discloses a determination to establish technical standards for field, laboratory, and classroom work in the *here's-the-way-to-do-that* style of characterization. Lack of confidence in the methods set forth is nowhere apparent. A guide to *savoir faire* for the clinician, a hint to the laboratory worker, and a suggestion to the pedagogue sum up the innermost intentions shown. When experience is verbalized, it rings the bell, whereas collections of printed indecisions hit with a plump. Moreover, the ambition to lift that segment from the sum of veterinary medical knowledge and set it apart as a special branch of study is truly commendable, since the advance of medicine has removed the mantle from the imperfections of physical diagnosis and brought in the "radar" of the laboratory to keep the special senses from blundering forever in a maze of uncertainties. In fact, clinical pathology is too precious a branch of learning to be scattered *ad libitum* through the literature by Tom, Dick, and Harry. It has foundations to standardize and to accept as do all branches of the medical sciences, which, up to the present time, have been hidden piecemeal in the writings on general subjects as if the diagnostic work, from patient or autopsy to the laboratory and back to the diagnostician, is but a fringe of veterinary medicine. Perhaps the name ought to be changed. "Clinical Pathology" signifies the bedside or, at most, "in the presence of the patient," whereas the science deserts that focus to do the detective work elsewhere—the investigational work that **apprehends** the criminal.

The reviewer is, however, obligated to tell the prospective reader that this book is an introduction to, not a complete coverage of, the subject implied by the title. The clinical pathology (in the accepted sense of the term) of all the diseases of the whole gamut of domestic animals would require a larger tome. As a matter of fact, the scope of the subject is well exposed on the 25 pages tabulating the "Data for the Differentiation of Diseases" of cattle, horses, sheep, hogs, poultry, and laboratory rodents. There are 194 laboratory problems with abridged hints on the handling of each. That is the foreseeable table of contents for the preoccupation of future writers on this fundamental branch of veterinary medicine. The author is entitled to the profound appreciation of the veterinary profession for mapping out the schedule of a new branch of study, as well as for the book's value in the routine work of veterinary practice.—[*Manual of Veterinary Clinical Pathology. By David L. Coffin, V.M.D., School of Veterinary Medicine, University of Pennsylvania. Cloth. 263 pages. Illustrated. 1 color plate. Comstock Publishing Company, Ithaca, N. Y. 1945. Price $4.00.*]

Mycotic Allergy

The allergy and resistance acquired in given experimental mycoses in guinea pigs were shown by inoculations of *Nocardia madurae*, which provoked an allergy detectable by intradermal injections of culture filtrate of the same fungus. The same effects, to a less marked degree, were provoked with *Sporotrichum biparasiticum*. The allergy was not strictly specific. Guinea pigs affected with experimental ringworm (*Ctenomyces mentagrophytes*) showed slight reaction to tuberculin, and those inoculated with BCG reacted to culture filterate of *N. madurae*, *Sp. biparasiticum*, or fungi of ringworm. A recent attack of ringworm did not protect against *N. madurae*, and guinea pigs inoculated with *Sp. biparasiticum*, or BCG, contracted a relatively benign *Ct. mentagrophytes* ringworm. Since various morphologic modifications were observed in cultures, the authors suggested that the fungi of ringworm in animals naturally infected may be subject to identical variations.—*Report of Pasteur Institute of Algeria, 1943.*

In these days, money paid to a veterinarian for examining and treating a herd of breeding cows is a good investment—such is the advice which E. J. Perry gives to dairymen of New Jersey.

THE NEWS

AVMA Activities

Fund to Be Raised for Veterinary Research

The American Veterinary Medical Association announces its intention to seek contributions for a fund to support research in veterinary science. President Farquharson has apppointed a special committee, under the chairmanship of the treasurer of the Association, to inaugurate a campaign at once. As its first task, the Committee hopes to collect a fund of at least $100,000 from members of the profession. It is expected that the profession will manifest confidence in the plan by making a substantial cash contribution before larger contributions are solicited from others. The Committee expects to approach animal lovers, livestock owners, and commercial organizations interested, for contributions toward a much larger sum. Contributions to this fund may properly be deducted when computing income taxes.

The objects of this fund are two-fold:

1) To advance knowledge in both the basic and the applied aspects of veterinary science.

2) To assist in the training of promising young scientists in these fields by affording them financial support, in the form of fellowships, for graduate education.

The funds obtained will be administered by the AVMA Research Council.* Applicants for fellowships supported by this fund must be citizens of the United States or Canada; they must have a veterinary degree from an institution approved by the American Veterinary Medical Association; they must have demonstrated ability, or promise of ability, in the field of their choice; and they must be accepted as graduate students by an educational institution approved for the purpose by the Research Council. These fellowships will not be limited to veterinary institutions. Fellows may work wherever facilities are adequate and competent supervision of their work is available.

Further details of the plan will be announced soon in the veterinary press and at veterinary meetings. In due time, all graduate veterinarians in North America will be approached individually for contributions, and it is hoped that they will respond liberally. The goal for this group may be attained, and even surpassed, by a minimum donation of ten dollars by each veterinarian.

So far as we know, such a fund has never been sought from any professional group. It is hoped that the profession will rise to the occasion and demonstrate to others that it has pride and faith in its work and an earnest desire to improve its services through wider knowledge of animal diseases and better training for its members. We cannot expect others to support such efforts with liberality unless they are impressed with our own seriousness of purpose.

Veterinarians are asked to give careful thought to this matter and be ready to respond when called upon to do so.

SPECIAL COMMITTEE ON FINANCING RESEARCH
s/ J. V. LACROIX, *Chairman*
C. C. HASTINGS
W. A. HAGAN

*Since it was established in 1941, the Research Council has outlined and approved projects in all of the major fields, but funds have been available to support only two of them.—Ed.

Board of Governors and Executive Board Meet

The Board of Governors met in Chicago on Dec. 1, 1945, and the Executive Board on December 2. In addition to routine matters, the following business was transacted.

The Board of Governors selected Boston for the 1946 annual meeting and the Executive Board set the week of August 18 for the sessions. The Hotel Statler will be headquarters and has confirmed the dates of August 18-23 for the meeting.

The Board received a report from the Special Committee on Food Hygiene embodying a basic food inspection plan. The report was approved for publication and distribution, in reprint form, to interested agencies and individuals. (The article appears in this issue.)

The chairman appointed a special committee of the Board to study the recommendations in President Farquharson's address at the 1945 business meeting, and to report their findings with respect to implementing the measures advocated.

The Executive Board elected the three members of the Executive Committee of the new Council on Education as required by the recent by-law amendment. Dr. W. A. Hagan was selected to represent the field of basic sciences; Dr. James Farquharson, the field of clinical sciences, and Dr. W. A. Aitken of Merrill, Iowa, the field of active general practice.

The Board authorized Associate Editor R. C. Klussendorf to make a study of the possibilities of a lay publication, and to submit full details at the next annual meeting.

The purchase of U. S. Treasury Bonds in the amount of $5,000 was authorized.

The appointment of a special committee to obtain funds for veterinary research, under the administration of the Research Council, was approved. The committee comprises Dr. J. V. Lacroix, *chairman*, and Drs. W. A. Hagan and C. C. Hastings. (See announcement elsewhere in this issue.)

Meetings of AVMA Committees

Several committees of the Association met in Chicago on or about Dec. 1, 1945, while their members were attending other sessions.

The Special Committee on Food Hygiene convened at the AVMA office on November 29-30. Those present were Dr. O. W. Seher, *chairman*, and Drs. M. O. Barnes, M. R. Clarkson, H. E. Kingman, Jr., and E. M. Lynn.

The Special Committee on Postwar Planning met for an all-day session on December 3. Present: Chairman H. L. Foust and Drs. J. A. Barger, Kenneth G. McKay, W. H. Riser, and Col. Seth C. Dildine.

The Research Council held an evening session on December 4. This was attended by Chairman E. T. Hallman, Secretary H. H. Dukes, and Drs. G. H. Hart, James Farquharson, W. F. Guard, C. F. Schlotthauer, R. A. Kelser, H. L. Foust, and M. A. Emmerson. Also present were President-elect B. T. Simms, Executive Secretary J. G. Hardenbergh, and Associate Editor R. C. Klussendorf.

Veterinary Scholarship Awards

The student achieving the highest average grade during the freshman, sophomore, and junior years of the regular curriculum at each of the ten veterinary colleges recognized by the AVMA will be eligible for a scholarship award of $300. The Borden Company Foundation, Inc. has made available to each school $1,500 to cover such an award in each of five years.

The pullet crop for 1945 was 23 per cent greater than that of 1944, and 10 per cent greater than the 5-year average.

APPLICATIONS

The listing of applicants conforms to the requirements of the administrative by-laws—Article X, Section 2.

First Listing

ANDERES, ROBERT L.
6501 Belinder Road, Kansas City 5, Mo.
D.V.M., Kansas State College, 1934.
Vouchers: J. L. Wells and A. H. Quin.

BROWN, JAMES R.
100 Jackson St., Demopolis, Ala.
D.V.M., St. Joseph Veterinary College, 1920.
Vouchers: B. N. Lauderdale and E. E. Williams.

BUNDE, HAROLD J.
Bowling Green, Mo.
B.V.Sc., Ontario Veterinary College, 1939.
Vouchers: J. L. Wells and L. E. Bodenwiser.

CHENG, CHING-TUAN.
National Research Bureau of Animal Industry, Nanking, China.
D.V.M., Michigan State College, 1943.
Vouchers: C. F. Clark and F. K. Thorp, Jr.

FARRELL, JOSEPH M. JR.
P. O. Box 65, Lindale, Texas.
D.V.M., Texas A. & M. College, 1944.
Vouchers: R. C. Dunn and A. A. Lenert.

SULLIVAN, J. RALPH.
232 Lee St., Montgomery, Ala.
D.V.M., Alabama Polytechnic Institute, 1918.
Vouchers: B. N. Lauderdale and E. E. Williams.

WEBSTER, JOHN S.
Schomberg, Ontario, Can.
B.V.Sc., Ontario Veterinary College, 1938.
Vouchers: H. S. MacDonald and W. Moynihan.

Second Listing

Arthur, Herbert E., P. O. Box 245, Auburn, Ala.
Cauthen, George E., Wright's Mill Road, Auburn, Ala.
Cook, Frank B., Box 177, Fort Deposit, Ala.
Dehaney, J. M., 88 Pine St., Mt. Holly, N. J.
Johnson, Robert A., 503 Montgomery St., Andalusia, Ala.
Jones, Benjamin F., Geneva, Ala.
Lee, Wilkie H., Robertsdale, Ala.
Miller, Maurice F., 33 Grow Ave., Montrose, Pa.
Milligan, John G., Marion Junction, Ala.
Parrish, Guy M., 409 Macon Ave., Louisville, Ky.
Summers, Mark E., Whitehall, Wis.
Swindle, B. Conwell, P. O. Box 377, Auburn, Ala.
Vogel, William M., 437 Sandhurst Drive, Dayton 5, Ohio.
Wesley, Joseph L., 610th AAF BU Sq. F., Eglin Field, Fla.

1945 Graduate Applicants

Second Listing

Alabama Polytechnic Institute

Hayman, William P., Jr., D.V.M., Box 711, Bartow, Fla.

Robinson, Leon K., D.V.M., Rt. No. 1, Box 66, Decatur, Ala.

Colorado A. & M. College

Collingson, Roger W., D.V.M., 707 York Rd., Towson, Md.

Ontario Veterinary College

Frank, Julius F., B.V.Sc., Macdonald College Ontario Veterinary College.

Texas A. & M. College

Dill, Clinton P., D.V.M., Box 191, Borger, Texas.

U. S. GOVERNMENT

Equine Encephalomyelitis.—A summary of cases, from Dr. B. T. Simms, chief, Bureau of Animal Industry, indicates that up to November 23, there had been reported 2,818 cases of equine encephalomyelitis this year, of which almost half (1,256) were reported after October 3. This compares with 9,816 reported in 1944 to Oct. 28, and substantiates the earlier estimate that this year's cases would total about one-third of the number reported last year.

AMONG THE STATES

Alabama

State Association.—The Alabama Veterinary Medical Association met at the Jefferson Davis Hotel, Montgomery, on Oct. 19, 1945. Mayor David E. Dunn presented an address of welcome, to which Dr. G. J. Phelps, Montgomery, responded. Dr. H. L. Allen, Demopolis, delivered his presidential address.

The scientific program included the following papers: Dr. B. T. Simms, Washington, D. C., "Parasites"; Dr. A. L. Holloway, Mobile, "Method of Handling Anthrax"; Dr. W. S. Bailey, Auburn, "Collecting, Preparing, and Shipping Laboratory Specimens"; Dr. L. E. Beckman, Tuscaloosa, "Clinical Diagnosis of Leptospirosis"; Hon. Joe N. Poole, commissioner of agriculture, Montgomery, "Future of the Livestock Industry in Alabama"; Harold N. Johnson, M. D., Montgomery, "Virus Diseases"; Dr. W. E. Cotton, Auburn, "Some Problems in Brucellosis"; Mr. J. K. Neel, Montgomery, "D.D.T."; Dr. H. F. Findley, Atmore, "Encephalomyelitis"; Dr. A. R. Gissendanner, Dothan, "Handling an Outbreak of Hog Cholera"; Dr. F. A. Clark, Montgomery, "Status of Meat Slaughtering and Meat Inspection in Alabama"; Dr. B. F. Cox, Auburn, led a discussion panel on penicillin.

Officers elected for the coming year are: W. W. Staples, Anniston, *president;* J. H. Milligan, Montgomery, *vice-president;* I. S. McAdory, Auburn, *secretary-treasurer.*

Alaska

Rabies Outbreak.—An epizoötic presumed to be rabies is reported from several areas of Alaska. The infected foxes have attacked human beings, dogs, and tires on moving automobiles.

California

Organize Harness Racing Association.—The spirit of the 1890's flared up in 1945, when the Western Racing Association was organized at Los Angeles. Among the directors are Gene Autry, well-known film star, Earl Gilmore, the celebrated cowboy actor, and Harry L. Warner, moving picture magnate. The president is Walter E. Smith and the secretary, Emmett "Pat" Doherty, of Los Angeles. The first meet will be held at Santa Anita in March, following the closing of the running races. In the heyday of "sulky racing," California bred many famous trotters and pacers.

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Personal.—Dr. N. Lew McBride announces the establishment of offices and hospital for the practice of veterinary medicine at 384 S. Raymond Ave., Pasadena 2.

Canada

Mortality from Tuberculosis Lowest on Record.—The Dominion Bureau of Statistics has released figures showing that the mortality from tuberculosis (human) in 1944 was the lowest ever recorded. The rate was lowest in Saskatchewan and highest in Quebec. The report shows that the 1944 death rate per 100,000 population was:

Saskatchewan	25.3
Ontario	26.9
Alberta	35.7
Manitoba	51.2
British Columbia	55.3
Nova Scotia	58.7
Quebec	75.0

The Dominion rate was 48.0, a reduction from 52.3 for 1943. Prince Edward Island showed an increase from 46.2 to 63.7, and New Brunswick from 48.6 to 51.7 over 1943.

Canal Zone

Association Meetings.—The Canal Zone Veterinary Medical Association, which was organized in 1944 and became affiliated with the AVMA the same year, holds regular meetings on the third Thursday of each month, alternating these meetings between the Atlantic and Pacific sides of the Isthmus.

Officers of the association are: Dr. C. C. Clay (Mich., '17), *president;* Dr. F. F. Dowd

(Corn., '10), *president-elect;* and Dr. Robert G. Matheney (Tex., '43), *secretary.*

Colorado

Fox and Mink Show.—Drs. B. R. McCrory, Webster Grove, Mo., and F. X. Gassner, Fort Collins, Colo., acted as veterinarians for the Mountain States Fox and Mink Show, held at Denver Nov. 17-20, 1945.

Florida

Personal.—Dr. J. Eric Anderson (A.P.I., '43) has established a practice at Delray Beach. He was formerly at Palatka.

Illinois

Bovine Mastitis Control Program.—The Department of Veterinary Pathology, University of Illinois, under whose directive a state-wide program of bovine mastitis control is being carried on, gives the following instructions on the collection of milk samples for microscopic examination:

1) Collect an 8 cc. (2 cc. from each quarter) composite sample into a sterile test tube, one containing preservative (sodium azide and brilliant green) if the sample cannot be incubated within twenty-four hours.

2) Collect the sample at least two hours after milking. Before taking samples, wash the udder and teats with chlorine solution, 200 to 400 p.p.m. Wash the hands in chlorine solution also.

3) Discard first two streams from each quarter into a strip cup.

4) Label all samples. Refrigerate the samples if not placed immediately into the incubator.

5) Incubate at 37 C. for twelve to fifteen hours.

6) Using a 4-mm. loop (24-gauge wire bent around a 6-penny wire nail), spread a loopful of well-mixed sample over a surface 4 by 8 mm. Allow smears to air dry; fix them by heating over a flame and then cooling or by placing slide in the incubator at 37 C. for an hour.

7) Stain slide by immersing in xylol for one minute and then in alcohol (95%) or rubbing alcohol (70%) for one minute. Drain off alcohol and immerse in methylene blue staining solution for about one minute. Examine with oil immersion lens.

8) Distinguish positive, suspicious, and negative cows by the number of coccal chains or clumps.

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Sanitary Association.—The United States Livestock Sanitary Association met at the La Salle Hotel, Chicago, December 5-7. In addition to the reports of committees, a variety of subjects was presented. The following veterinarians read papers: B. T. Simms, James H. Steele, A. R. Miller, Benjamin Schwartz, and A. E. Wight, all of Washington, D. C.; J. M. Murphy, Sussex, N. J.; F. R. Beaudette, New Brunswick, N. J.; R. Fenstermacher, St. Paul, Minn.; C. C. Morill, Urbana, Ill.; A. L. Brueckner, College Park, Md.; Alexander Zeissig, Ithaca, N. Y.; Howard W. Johnson, Auburn, Ala.; Lieut. Col. R. W. Rushmore, U. S. Army; A. B. Crawford, Beltsville, Md.; Asa Winters, Albany, N. Y.; and Glenn C. Holm, Moscow, Idaho. Other papers were read by Prof. K. C. Seeger, Georgetown, Del.; Harold N. Johnson, M. D., Montgomery, Ala.; Wm. C. Reeves, Ph. D., San Francisco, Calif.; Mr. R. M. Core, Franklin, Ind.; and Carl F. Jordan, M. D., Des Moines, Iowa.

All sessions were well attended, and the registration was gratifying.

Officers elected for the coming year are: William Moore, Raleigh, N. Car., *president;* Will J. Miller, Topeka, Kan., *first vice-president;* R. A. Hendershott, Trenton, N. J., *secretary-treasurer.* The fiftieth annual meeting will be held Dec. 4-6, 1946.

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Northwestern University Employs Veterinarian.—Dr. Roger P. Link (I.S.C., '34), has accepted a position as assistant professor of physiology in the Medical School of Northwestern University, Chicago. Dr. Link leaves his work at Kansas State College, Department of Physiology, to take charge of the new, enlarged, and thoroughly modern quarters for experimental animals at Northwestern. He will do research work and teaching.

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Veterinarian in the Meat Industry.—Dr. H. E. Kingman, Jr. (Colo., '33) has provided us with advance copy of an advertisement sponsored by Wilson and Co. for publication in twelve midwestern agricultural papers. It introduces the U. S. meat inspector as "a friend you may never see."

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Chicago Association.—An overflow gathering of members and guests attended the December meeting which was held one week earlier than usual. Dr. Andrew L. MacNabb, principal of the Ontario Veterinary College, spoke on "Veterinary Education in Canada," and Dr. F. W. Schofield, also from Guelph, discussed "Penicillin in the Treatment of Mastitis."

The ladies' auxiliary held a Christmas party the same evening, Dec. 4, 1945.

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Pulling Contest Record.—A Belgian team, weighing 2,900 lb., lifted 3,025 lb. for 27.5 ft. on the dynamometer, to set a new record for light weight teams in Illinois. The record breaking team is owned by Short Newman, of Oblong (Crawford county).

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Fixation Bone Pin.—A new, improved, external fixation bone pin, which consists of a combined pilot-type drill and reamer, is now

being distributed for use with the Stader fracture splint solely through General Electric X-Ray Corporation. The drilling of cortical bone is greatly facilitated because the reamer makes it possible to produce a hole of the exact diameter of the pin shaft.

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Graham Appointed Dean at Illinois.—Dr. Robert Graham has been named dean of the University of Illinois' new college of veterinary medicine by the board of trustees. Dr. Graham

Dr. Robert Graham

has been head of the department of animal pathology and hygiene and was the unanimous choice of the committee appointed to make recommendations for the new position.

Born at Ames, Iowa, Dr. Graham was awarded his D. V. M. degree by Iowa State College in 1910. He was professor of veterinary science at the University of Kentucky from 1911 to 1917, when he joined the faculty of the University of Illinois. He was made head of his department in 1941.

A national authority in the field of animal pathology, he developed the first botulinus antitoxin in 1919 and has made significant and outstanding contributions to our knowledge of forage poisoning and sleeping sickness in horses, brucellosis and mastitis in cattle, erysipelas and enteritis in swine, parasitism and abortion in sheep, and vaccination of chickens and turkeys to immunize them against fowl pox.

Dr. Graham was an officer of the Veterinary Corps in World War I, and was in charge of the regional veterinary laboratory at Atlanta and Fort McPherson, Ga.

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Fat Stock Show.—The Chicago Market Fat Stock Show, of 1945, wartime substitute of the International Livestock Exposition, attracted more nation-wide attention than usual on account of the deluge of fine stock exhibited and the publicity given to the 4-H Clubs in the newspapers throughout the country. The 4-H boys and girls stole the show and also most of the hotel rooms. Published interviews with the 4-H members appear to indicate that few of them expect to remain on the farm. Answer that one.

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Horse and Mule Association Meets.—The 26th annual meeting of the Horse and Mule Association of America, held at Chicago on Dec. 5, 1945, was attended by representatives from more than 20 states, which indicates that there still is a lively interest in the approximately 13 million horses and mules in the United States.

Brig. Gen. R. A. Kelser, director of the Veterinary Division, Office of The Surgeon General, United States Army, spoke to the assembled members about periodic ophthalmia, or moon blindness. He reported that at Front Royal Remount Station, 109 of every 1,000 horses received had developed periodic ophthalmia until the feeding of crystalline riboflavin at the rate of 40 mg. daily, was begun. Not one horse so treated developed the condition in the Remount.

Mr. Erwin Dygert reported that 10,300 horses and 300 mules had been purchased for UNRRA, and that most of them had been exported to southern Europe. In addition, about 15,000 animals (mostly mules) owned by the U. S. Army in Europe have been declared surplus and have been turned over to UNRRA for distribution in Greece, Jugoslavia, Czechoslovakia, and Poland.

Col. Thomas Johnson discussed the military use of horses and the need for them in South America, where he is stationed.

President Louis E. Stoddard, of New York; Vice Presidents C. J. Lynn, of Carmel, Ind., and Ira Dryman, of Lexington, Ky.; and Executive Secretary Wayne Dinsmore will serve another year in their respective offices. F. L. Morrow, of New Britain, Conn., was elected treasurer.

Indiana

Personal.—Dr. Harold D. James (O.S.U., '39) has been retired from active duty as a captain in the Veterinary Corps. He has resumed his duties in swine disease research under Dr. L. P. Doyle, at Purdue University.

Iowa

North Central Association.—The Warden Hotel, Fort Dodge, was the scene of the meeting of the North Central Iowa Veterinary Medical Association on October 31, the first to be held since the spring of 1942. The following diversified program was presented: Dr. H. C. Smith, Sioux City, discussed the use of the sulfonamides in the treatment of pneumonia in cattle, sheep, swine, and horses. Discussion was continued from the floor by Drs. F. H. Kelly, Goldfield, and John Dewar, Cherokee. Drs. T. S. Leith, Ames, and H. E. Pinkerton, Fort Dodge, then demonstrated the technique of bleeding swine from the vena cava and also from the ear vein. Drs. P. O. Dorweiler, West Bend; S. D. Linn, Humboldt; C. L. Telleen, Gowrie; and V. R. Howie, Manson, talked on poultry diseases. They were pinch hitting for Dr. C. D. Lee, Ames, who had been scheduled to speak on this subject but was unable to do so because of a fractured leg. Mr. S. L. Bickal, Fort Dodge, read a paper entitled "Pharmaceutical Products"; additional comments on this subject were made from the floor by Drs. E. R. Truax, Sac City, and O. N. Emerson, Eagle Grove.

The ninety veterinarians attending elected the following officers: L. W. Schalk, Alden, *president;* O. N. Emerson, Eagle Grove, *president-elect;* B. J. Gray, Fort Dodge, *secretary-treasurer;* S. D. Linn, Humboldt; C. J. Mickelson, Webster City; and M. H. Carter, Thor, members of the executive board.

s/ B. J. GRAY, *Secretary.*

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The Steer of the Year.—When Carl Henkel, of Mason City, and Joseph Duea, of Belmond, sold Tomahawk, grand champion of the Chicago Market Fat Stock Show, in December for $11,100 to the Henrici restaurant, a new record was established for the price of a steer. Tomahawk, a Shorthorn, weighed 1,100 lb., making the selling price $1,000 per cwt. which, calculated on the basis of dressed weight, is $20 per pound of beef. Computed from the prices on the Henrici menu, the total return will be but $500 over the cashier's desk. The advertising value is not that easy to put into figures.

At the Eastern States Livestock Exposition in 1933, the grand champion steer sold for $11.15 per pound, but weighing 915 lb., the sale brought the owner only $10,202.

Kansas

District II.—Members of the second district and their wives met at the home of Dr. and Mrs. Jay Reynolds, Great Bend, on November 17. Dr. A. H. Quin, Kansas City, Mo., enumerated some of the newer antibiotics and elaborated on the actions of penicillin. He particularly drew attention to the use of penicillin in navel ill, peritonitis, ophthalmia, and mastitis.

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Anaplasmosis Treatment.—Dr. T. R. Allison, Winfield, reports recovery of all cases of anaplasmosis treated with penicillin. Dairy cows, in which a diagnosis of anaplasmosis was confirmed by laboratory examination, were treated with 100,000 units of penicillin, which was repeated in twenty-four hours. Dr. Allison says: "They made a quick recovery and were back in full milk production in a very short time."

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Undulant Fever.—Drs. George J. Kiger, Alta Vista, and M. G. Enlow, Pratt, have suffered attacks of undulant fever, but are reported making favorable progress.

Louisiana

Healthiest 4-H Member.—John Harold Savoy, a student at Creole high school, Cameron parish, was named the nation's healthiest 4-H club member at the national congress in Chicago. His home is in a definitely rural section, where coastal marshlands abound and 4-H activities are limited. He has been a club member for seven years, and his selection for one of the most coveted awards of the annual congress is the result of following good health practices for many years.

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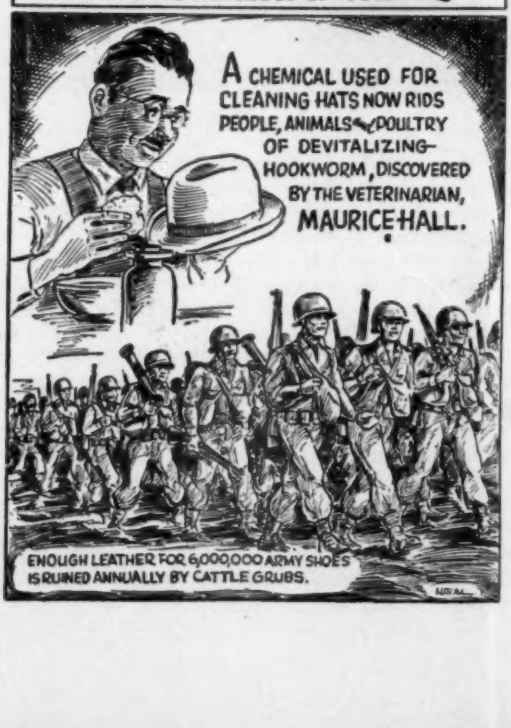
Brahman Show.—The first National Brahman Show will be held at Baton Rouge, February 12-17, in conjunction with the Louisiana Adult Livestock Show. Brahmans will be judged by Frank Scofield, Austin, Tex., and premiums amounting to $3,940 will be awarded to outstanding animals.

LIVESTOCK HEALTH ODDITIES



Massachusetts

State Association.—The Massachusetts Veterinary Medical Association met at Boston, December 19. Following the dinner, Dr. F. M. Austin, president of the association, showed a colored picture with sound effects on the breeding, schooling, and showing of saddle horses.

s/ H. W. JAKEMAN, *Secretary.*

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The American Milk Goat Record Association.—The forty-first annual meeting was held at Worcester, Oct. 22, 1945, under the presidency of Dr. A. J. Durant, head of the Department of Veterinary Science, University of Missouri, and an ambitious leader of the milk goat industry of the United States. In the spirit of comradeship and general interest in that branch of husbandry, Dr. L. A. Paquin, of Webster, greeted President Durant and delegates in behalf of the Massachusetts Veterinary Medical Association and the AVMA. Thus, two of the world's oldest veterinary organizations paid quiet tribute to a colleague distinguished for leadership in a growing branch of the livestock industry.

The AMGRA membership of 1,252 (a gain of 37 per cent in the last twelve months) and the issuance of 300 Star Certificates during the year, tell the story of capriculture in this country. The association conducts a comprehensive system of Dairy Herd Improvement and Advanced Registry, based upon both milk and butterfat testing for both short and long periods. The short period is for those who do not find it feasible to arrange for long-time testing.

The association is organized like the AVMA. The United States and Canada are divided into six districts, each of which is represented by three directors who are elected annually. The annual meetings in the odd years are held in outlying districts and in the even years in the central district. Thus, the 1946 meeting will be held about the middle of October, at Columbia, Mo., home of President Durant.

s/ L. A. PAQUIN

Michigan

State Association.—The Michigan State Veterinary Medical Association which was founded in 1883, heard and discussed at length a paper on bovine tuberculosis by J. A. Dell, V.S., of Ann Arbor, at its second annual meeting in East Saginaw, Feb. 3, 1884, or two years after Koch's discovery of the tubercle bacillus. Was this a "first"?—*From J. Comp. Med. and Surg., 5, (Apr. 1884): 189.*

Minnesota

France Honors Major Macy.—Major Harold Macy, professor of dairy bacteriology, University of Minnesota, was named Chevalier of the Legion of Honor, by the French Government for distinguished service in the course of the war. He was cited as "An eminent bacteriologist who, during the course of the war, never ceased to show in a most affectionate manner his friendship for France." In London, Major Macy was head of the Medical Mission of Civil affairs.

Mississippi

Livestock Auctions.—Data collected on the operation of 32 out of 37 livestock markets in the state, in 1944, regarding prices, personnel, installation, operation, and the spreading of diseases, are presented by D. W. Parvin (Mississippi Agric. Exper. Sta. Bull. 400, (1944): 87.) The livestock sold numbered 800,000 head, valued at $20 million. A questionnaire circulated among farmers indicates that more livestock would be consigned to the auction if the charges were reduced, and steps were taken to prevent the spread of diseases. Higher prices and convenience were cited as the chief reasons for selling through auctions.

Missouri

Kansas City Association.—The work and projects of the National Livestock Loss Prevention Board under the title, "An Ounce of Prevention," was presented at the November (1945) meeting by R. L. Cuff, Kansas City livestock commissioner, and Fred Olander, chairman of the NLLPB, in the form of a multicolored sound film depicting losses suffered in the handling and shipping of livestock.

s/ GAIL B. SMITH, *Secretary.*

• • •

Delegate to House of Representatives.—At the conference for veterinarians at Columbia, the members assembled elected Dr. H. E. Curry, Jefferson City, as delegate to the house of representatives, with Dr. Fred L. Seevers, Pleasant Hill, as alternate. Dr. Glen L. Dunlap was elected resident secretary of the AVMA.

s/ J. L. WELLS, *Secretary.*

Montana

Barley Ergot Causes Pig Losses.—Experiments of Drs. Nordskog and Clark, of the state agricultural station, showed that 9 pregnant sows fed barley containing 0.5 to 1.0 per cent of ergot farrowed but 38 live pigs out of a total of 83 born, compared with 12 control sows which farrowed 106 pigs of which but 3 were born dead. The study further showed that ergot prevents udder development and milk secretion in addition to fetal deaths. In one small area, 500 pigs died of starvation in one to ten days.

Nebraska

Quintuplet Calves.—Five viable calves, 4 males and 1 female, born to a crossbred Hereford-Shorthorn cow on the farm of Lee

Schmoldt, near Fairbury, Nov. 20, 1945, were alive and thriving at the age of 1 week, according to a news dispatch to the *Chicago Tribune*, November 27. The history of this pregnancy and parturition from the local veterinarian is awaited for the records.

New York

New York City Association.—Meetings of this association are now being held at Hotel Pennsylvania, Conference Room No. 2. At the meeting held on the evening of December 5, Dr. J. A. S. Millar, Deal, N. J., spoke on the subject, "Tumors in Dogs."

All incumbent officers were unanimously reelected: Dr. E. R. Cushing, Plainfield, N. J., *president;* Dr. Leonard Goss, New York, *vice president;* Dr. C. R. Schroeder, Pearl River, *secretary-treasurer.*

s/ C. R. SCHROEDER, *Secretary.*

• • •

Cornell Revises Veterinary Curriculum.—New York State Veterinary College has just released an announcement which indicates that a thorough study and a careful revision of the veterinary curriculum has been undertaken, although it is stated that "the curriculum of the College is in a transitional state."

New Curriculum

Subjects	Credits	
First year	1st term	2nd term
Anatomy	7	7
Histology & Embryology	4	4
Organic Chemistry	5	
Physiological Chemistry		6
Animal Husbandry	3	
Physiology		3
Second Year	1st term	2nd term
Physiology	3	
Experimental Physiology	3	
Bacteriology and Immunology	9	
General Pathology	4	
Special Pathology		5
Genetics		3
Therapeutics and Pharmacy		6
Parasitology		4
Animal Feeds and Nutrition		4
Third Year	1st term	2nd term
Food Quality Control	6	
General Surgery	4	
Surgical Exercises	1	1
Infectious Diseases	3	
Diseases of Large Animals	5	3
Diseases of Small Animals	3	
Poisonous Plants	1	
Applied Anatomy	1	1
Clinical Orientation	Cr.	Cr.
Obstetrics		5
Special Surgery		5
Diseases of Poultry		3
Roentgenology		1
Parasitology		1
Fourth Year	1st term	2nd term
Diseases of Large Animals	2	4
Diseases of Small Animals	3	
Clinical Conferences	Cr.	Cr.
Clinics	Cr.	Cr.

Clinics will be held all day, Monday through Friday, beginning at 9 a. m. and on Saturday until 1 p. m. This work will be given coöperatively by several departments, among which students will divide their time. Students will be assigned in small groups to special work in the surgical and small animal clinics, the diagnostic laboratories, and in topographic anatomy, blood and urine chemistry, parasitology, hematology, clinical pathology, bacteriology, and serology. In the ambulatory or out-clinic which will be conducted, students will have an opportunity to observe such diseased farm and dairy animals as cannot be entered in the clinics of the College, and to assist in handling cases in the same manner and in the same environment as are required of the country practitioner.

Ohio

Personal.—Dr. R. Forbes Colgate (Ont., '41) has been engaged by Dr. N. B. Tenille to act as his assistant in the West Toledo Animal Hospital, 3165 Sylvania Ave., Toledo.

Pennsylvania

Light Harness-Horse Sale.—The sale of 400 head of Standardbred horses at York, Oct. 24, 1945, is reported in *Harness Horse* as a "record breaker." The sale totaled $502,000, or an average of more than $1,000 per head. The top price was $11,500 and the lowest $50. The 73 head of the Hanover Shoe Farms brought $168,575.

• • •

Kelser Appointed Dean at Pennsylvania.—Brig. Gen. Raymond A. Kelser will retire about Jan. 15, 1946 after 27 years of service in the Veterinary Corps, U. S. Army, and has accepted the deanship in the School of Veterinary Medicine, University of Pennsylvania, according to a recent announcement by university officials.

Born at Washington, D. C., in 1892, Gen. Kelser received his D.V.M. degree from George Washington University in 1914, and an M.A. and a Ph.D. degree from American University in 1922 and 1923, respectively. He was bac-

teriologist for H. K. Mulford & Co. in 1914 and then joined the Pathological Division, Bureau of Animal Industry. Entering the Army Veterinary Corps in 1918, he helped to organize and later commanded the first army veterinary laboratory. From 1921 to 1925, and again from 1928 to 1933, he was in charge of the veterinary laboratory division of the Army Medical School in Washington, with an intervening period of service in the Philippines where he was a member of the U. S. Medical Department Research Board. Later, he was a research fellow in bacteriology at Harvard Medical School under Prof. Hans Zinsser. In 1938, he was made chief of the Veterinary Division, Surgeon General's Office, and was continued in this post, after the outbreak of World War II, until retired.

Brig. Gen. R. A. Kelser

General Kelser has made numerous contributions to scientific veterinary medicine and the control of animal diseases, including the development of a successful vaccine against rinderpest, the discovery of mosquito transmission of the virus of equine encephalomyelitis, studies on rabies and other filterable viruses, and Chagas disease; he is also the author of the textbook, "Manual of Veterinary Bacteriology." In 1942, he was awarded the International Veterinary Congress Prize by the AVMA.

• • •

Keystone Veterinary Medical Association.—The members met on November 28 to hear Dr. W. J. Lentz, Philadelphia, deliver an address on diseases of the eye, and to hear Mr. W. H. Colburn explain group accident and disability insurance for the veterinarian.

s/ R. C. SNYDER, *Secretary.*

Texas

State Association Moves Offices Again.—Circumstances necessitated moving the offices to rooms above the Aggieland Pharmacy, at the North Gate of the College. Mail may be addressed to Box 951, College Station, Texas.

s/ E. A. GRIST, *Editor* and *Secretary.*

• • •

Valley Veterinary Association.—The monthly meeting of the Association was held October 14, in Harlingen, and was devoted to the consideration of matters affecting dairy folks in the Rio Grande valley. The following day, with Dr. R. E. Carroll, president of the Valley Association, acting as master of ceremonies, the film "Science of Milk Production" (Purina Mills) was shown to a group of about 175 dairymen as part of a dairy day. Following the picture, Drs. Brock and Grist presented the problems of brucellosis and mastitis to the group and answered questions.

• • •

San Antonio Association.—Some 28 members of the San Antonio Veterinary Medical Association met on October 18 to participate in a general consideration of the future of veterinary medicine, with special reference to educational channels. Dr. W. G. Brock acted as moderator for what amounted to a forum of all the members present.

• • •

Junior AVMA.—Dr. J. D. Williams (Tex., '35) Colorado City, recently spoke to members of the Junior Chapter of the AVMA on "Problems Affecting a Large Animal Practitioner."

• • •

New State Veterinarian.—Dr. Dan J. Anderson (Tex., '38) has been released from military service as a captain in the Veterinary Corps to assume the duties of his new office as state veterinarian.

• • •

Son Dies in Prison Camp.—Dr. and Mrs. D. C. Becker, Brenham, received the sad word that their son, Lt. Darwin C. Becker, had died in a Japanese prison camp Jan. 30, 1945. Delay in receiving word until now is the fault of the Japanese government. Dr. Becker was formerly a BAI Inspector.

Washington

State Association.—The Washington State Veterinary Medical Association held its annual meeting at Yakima on November 16 and 17. Lieut. Col. Leighton Bailey, Major G. C. Folger, and Capt. Dell Finch recounted some of their army experiences to highlight the first day's

meeting. Dr. E. A. Elmer, Seattle, reported on external fixation of fractures in small animals, and Dr. Ed Crook, Pullman, reported on the activities of the newly organized mastitis research and control program. Dean E. E. Wegner, Pullman, acted as toastmaster at the banquet.

Officers elected for the coming year are: Lieut. Col. Leighton Bailey, Spokane, *president;* Major G. C. Folger, Mount Vernon, *vice-president;* Dr. M. O. Barnes, Olympia, *secretary-treasurer.* Dr. Barnes was also elected delegate to the House of Representatives, with Dr. H. A. Trippeer, Walla Walla, alternate. Seven new members of the board of trustees were elected and installed.

s/ M. O. BARNES, *Secretary.*

Wisconsin

Death of John Letham.—The death of John Letham at Lake Geneva, Oct. 18, 1945, at the age of 86, removes one of the foremost figures from the Hereford field. He was an outstanding authority on the breed, and a famed writer on the *American Hereford Journal* staff for many years. Few are aware that Mr. Letham entered the American Veterinary College, New York, soon after his arrival from Scotland in 1870. His practical writing and sound philosophy are credited with having lent much to the development of the Hereford in this country. Obviously, much is owed to his veterinary studies at the dawn of his career.

• • •

Brucella Legislation.—To qualify as an "official vaccinate" a calf must meet five conditions: (1) vaccinated by approved veterinarian, (2) vaccinated with approved vaccine, (3) vaccinated between the ages of 4 and 8 months, (4) permanently identified with tattoo and pass tag in right ear, and (5) reported to the Wisconsin State Department of Agriculture in five days.

When vaccinated as an adult, the animal is identified by a triangular hole, measuring at least one-half inch on each side and punched in the *right* ear, and a pass tag with a serial number. Unvaccinated reactors are identified by a triangular hole punched in the *left* ear and an official reactor tag in the left ear.

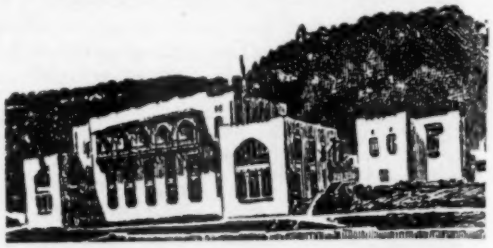
There is a provision for sale or movement, under permit, of reacting or adult vaccinated animals into herds having reactors.

A booklet of information has been issued as Bulletin 263 of the Wisconsin State Department of Agriculture, and a copy may be obtained from Dr. V. S. Larson, chief, Livestock Sanitation, State Capitol, Madison.

Foreign

Algeria

An example of medico-veterinary coöperation to the same end—human welfare—is seen in the functions of the Institute Pasteur of Algeria and its branches in Sahara and Morocco. To the advantage of its scientific researches—its principal objective—the Institute partici-

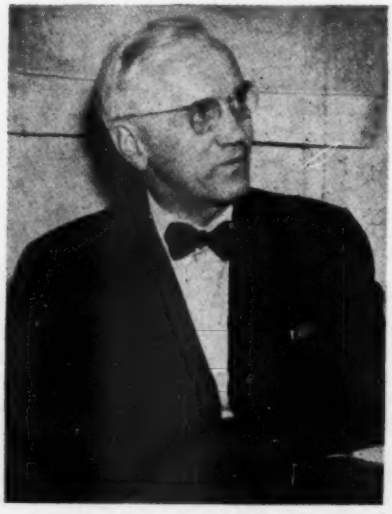


—*From Report of Pasteur Institute of Algeria*
Facade of the Pasteur Institute of Algeria

pates actively and materially in controlling ubiquitous and exotic diseases of man and animals. The report for 1944 contains comprehensive material on malaria, typhus, yellow fever, rinderpest, rabies, hog cholera, bovine piroplasmosis and theileriasis, smallpox, sleeping sickness, leishmaniasis, plague, cholera, tuberculosis, antiscorpio and antiviper treatments, diseases of dromedaries, mycoses, brucellosis, typhoid and paratyphoid fevers, vaccine and serum production, analyses for physicians, veterinarians, and farmers—a combination of services and morbid states inseparable in a general plan to serve mankind.

Great Britain

Award of Nobel Prize in Medicine.—Sir Alexander Fleming, discoverer of penicillin, was awarded the Nobel prize in physiology and



—*From Medical Times*
Sir Alexander Fleming

medicine for 1945. Sharing in the award were Sir Howard W. Florey and Dr. Ernest B. Chain, who fostered its development to an available life-saving remedy.

COMING MEETINGS

Kansas Veterinary Medical Association. Hotel Jayhawk, Topeka, Kan., Jan. 14-15, 1946. C. W. Bower, 3119 Stafford St., Topeka, Kan., secretary-treasurer.

Mississippi State Veterinary Medical Association. Hotel Edwards, Jackson, Miss., Jan. 14-15, 1946. G. D. Gates, Clarksdale, Miss., secretary.

Texas Veterinary Medical Association. Texas Hotel, Fort Worth, Texas, Jan. 14-16, 1946. E. A. Grist, Box 951, College Station, Texas, secretary.

California State Veterinary Medical Association. California Polytechnic School, San Luis Obispo, Calif., Jan. 15-17, 1946. F. P. Wilcox, 4219 Kenwood Ave., Los Angeles 37, Calif., secretary.

Louisiana State University and Louisiana Veterinary Medical Association. Annual Short Course. Louisiana State University, Baton Rouge, Jan. 16-17, 1946. A. H. Groth, Department of Veterinary Science, Louisiana State University, Baton Rouge, head.

Illinois State Veterinary Medical Association. Leland Hotel, Springfield, Ill., Jan. 17-18, 1946. C. C. Hastings, Williamsville, Ill., secretary-treasurer.

Iowa Veterinary Medical Association. Hotel Fort Des Moines, Des Moines, Iowa, Jan. 22-24, 1946. C. C. Franks, 720 Grand Ave., Des Moines, Iowa, secretary.

Michigan State College. Annual Conference for Veterinarians. School of Veterinary Medicine, Michigan State College, East Lansing, Jan. 23-24, 1946. Ward Giltner, School of Veterinary Medicine, dean.

North Carolina State College. North Carolina Conference for Veterinarians, 205 Polk Hall, Raleigh, N. C., Jan. 22-25, 1946. C. D. Grinnells, State College Station, Raleigh, N. C., program chairman.

Minnesota State Veterinary Medical Society. Hotel Nicollet, Minneapolis, Minn., Jan. 28-30, 1946. H. C. H. Kernkamp, University Farm, St. Paul 8, Minn., secretary.

Virginia State Veterinary Medical Association. John Marshall Hotel, Richmond, Va., Jan. 28-30, 1946. R. D. Hatch, Department of Biology, Virginia Polytechnic Institute, Blacksburg, Va.

Wisconsin Veterinary Medical Association. Park Hotel, Madison, Wis., Jan. 30-31, and Feb. 1, 1946. B. A. Beach, University of Wisconsin, Madison 6, Wis., secretary.

Kentucky Veterinary Medical Association. Kentucky Agricultural Experiment Station, Lexington, Ky., Jan. 31, and Feb. 1-2, 1946. F. M. Kearns, 3622 Frankfort Ave., Louisville 7, Ky., secretary-treasurer.

Connecticut Veterinary Medical Association. Bond Hotel, Hartford, Conn., Feb. 6, 1946. G. E. Corwin, 36 Capitol Ave., Hartford 6, Conn.

Alabama Polytechnic Institute. Annual Short Course for Veterinarians. Auburn, Ala., Feb. 26-28, 1946. E. S. Winters, College of Veterinary Medicine, administrative secretary.

DEATHS

Edward D. Criswell, (West., '04), 61, King City, Mo., died recently. Dr. Criswell was a member of the AVMA.

Lloyd Donnell (Ind., '18), 49, Chicago, Ill., was killed in an airplane crash at Yreka, Calif., Nov. 16, 1945. Dr. Donnell was a native of Clinton county, Ind., and a former well-known veterinarian of that locality. Four years ago, he received an appointment as a government meat inspector with headquarters at the Union Stockyards, Chicago. Recently he was transferred to Montague, Calif. Dr. Donnell was admitted to the AVMA in 1919.

John J. Hayes (N. Y. Amer., '00), Chicago, Ill., died recently. Dr. Hayes was admitted to the AVMA in 1913.

Bernard D. Kahl (M.S.C., '39), 30, Hamilton, N. Y., died several months ago. Dr. Kahl was admitted to the AVMA in 1939.

Samuel B. Moon (C.V.C., '07), 74, Rock Rapids, Ia., died on Oct. 29, 1945. Dr. Moon had been a member of the AVMA since 1924.

Trajan A. Shipley (C.V.C., '90), 81, Uvalde, Tex., died Nov. 24, 1945. Dr. Shipley was appointed assistant inspector at Chicago in 1895. He served in this capacity at Cudahy, Wis., and Cedar Rapids, Ia. In 1896 he was promoted to inspector, which position he held until his retirement in 1933. Dr. Shipley had been a member of the AVMA since 1910.

Jesse L. Slankard (St. Jos., '21), 63, Elk City, Okla., died Nov. 9, 1945. Dr. Slankard has carried on a general practice in Elk City since 1922.

M. E. Spratlin, Littleton, Colo., was killed in an auto accident Oct. 30, 1945. Dr. Spratlin had carried on a practice in Littleton for many years.

Abraham Travis, 73, Litchfield, Ill., died Nov. 14, 1945. Dr. Travis had practiced in Litchfield for the past forty-six years.

George C. Webb (Ont., '03), 67, Kewaunee, Wis., died Oct. 15, 1945. Dr. Webb served the Kewaunee area for some thirty-eight years and enjoyed the highest confidence and respect of those he served. He was admitted to the AVMA in 1942.

THE VETERINARY PROFESSION AND THE WAR

Release Points Reduced

Information just released indicates that the number of points necessary for discharge of officers from the Veterinary Corps has been reduced from 70 to 65. The age limit remains 42, and the active duty date prior to Dec. 7, 1941.

Colonel Seymour to Manila

After serving on Okinawa through the campaign and until the end of the War, Col. R. T. Seymour, V.C. (K.C.V.C., '15) was transferred to Manila and is now attached to Headquarters, AFWESPAC, Office of the Chief Surgeon, APO 707, San Francisco, Calif.

Meritorious Service Unit Plaque

The veterinary detachment, consisting of 18 officers and 25 enlisted men, of the California Quartermaster Depot, Oakland, has been awarded the Meritorious Service Unit Plaque for superior performance and outstanding devotion to duty in the inspection of subsistence of animal and seafood origin.

Veterinary Unit Twice Decorated

Capt. Costas A. Alvanos, V. C. (M.S.C., '39) is commanding officer of the veterinary company of the 10th Mountain Medical Battalion of the 10th Mountain Division, and he is assisted by Capt. Jack LaMont, V. C. (K.S.C., '43). This company was responsible for the care and treatment of about 900 mules and 150 horses prior to departure for overseas, and about half that number while in Italy. Their first aid station at the front dressed shrapnel wounds on captured enemy horses, as well as on Italian and U. S. mules and horses.

The two officers and the nine enlisted men were awarded the Combat Medical Badge for close support of the infantrymen and the Bronze Star Medal for meritorious achievement in action in the North Appenine, Po Valley, and Alps Mountains offensives.

Health of War Dogs

The Veterinary Division of the Army Medical Department has maintained the health of army war dogs at an excellent level. The outstanding achievements of war dogs on the field of battle have more than justified the time and expense involved in training and caring for them. To date, individual dogs and infantry dog platoons have received a total of twenty-two citations for outstanding service in the line of duty.

Veterinary Officer Decorated

Col. Ralph W. Mohri, V.C., of Manhattan, Kan., has been awarded the Legion of Merit for his work in the India-Burma theater. The citation says:

—U. S. Army Signal Corps
Colonel Ralph W. Mohri

"He was directly responsible for organizing and supervising the veterinary service of this theater . . . . he successfully inaugurated methods of animal disease control, particularly surra, whereby animal transport elements . . . . were maintained at maximum effectiveness." Colonel Mohri graduated from Kansas State College in 1929 and is a member of the AVMA.

Legion of Merit Awards

For exceptionally meritorious conduct in the performance of outstanding services, the Legion of Merit was awarded by the Adjutant General's department to:

Colonel Russel McNellis
Colonel Gerald W. Fitz

Veterinary Corps Officers Separated

The following names of Veterinary Corps officers, reported as separated since V-E Day, were furnished by Col. J. F. Crosby, Veterinary Division, Office of the Surgeon General, and supplement the list published in the December JOURNAL.

Alabama
Faulk, Archie L.

Arkansas
Reineccius, Jake L.

California
Arburua, Joseph M.
Hurt, Ross H.
Niemeyer, Wm. E.
Priddy, Charles W.

Colorado
Herzberger, A. C.
Milliken, John C.
Howarth, William A.

Connecticut
Anderson, James G.
Bushnell, Fred F.

Florida
Christian, Arthur B.
Lord, Willys E.

Georgia
Stewart, Abe Lincoln

Illinois
Brinker, Wade O.
Metz, Clark A.

Indiana
Burns, Kenneth F.
Kucher, Paul C.
Fly, Glen O.

Iowa
Bond, Thomas L.
Butler, Elliott A.
Geick, Harold L.
Linn, Frank J.
Storm, Robert E.
Ward, Donald E.

Kansas
Bruce, Ralph A.
Duncan, Glenn E.
Hein, Hubert R.
Watson, Bruce C.
Loyd, Paul T.
Maichel, George B.
Spencer, Ralph N.

Kentucky
Boyd, Wayne W.

Louisiana
Bowers, Grafton D.
Bryson, Bernard G.

Maine
Kaskin, Samuel T.

Maryland
Dinkel, John H.
Kerr, Virgil Milo
Richman, Silas
Rohrer, Raymond R.

Massachusetts
Boardman, William

Michigan
Court, Maurice J.
Waddell, Wm. F.
Walters, Maurice K.
White, Edward S.

Minnesota
Enge, Percy C.
Peterson, Alfred
Karlson, Alfred G.

Missouri
Callaway, Hugh P.
Millenbruck, E. W.
Poley, Linn S.
Shelby, Clarence F.

New Jersey
Shomer, Robert R.

New Mexico
Moore, Andrew A.

New York
Sherwood, W. J.
Whitehead, R. G.
Stieber, Sam

Nebraska
Duey, Fernley W.
Johnson, John C.
Price, Willet J.
Smith, Edwin J.

Nevada
Key, Joseph B.

Ohio
Folsom, Robert H.
Harris, William K.
Herman, Leslie F.
Hinkle, Truman B.
Hook, C. Johnson
James, Harold D.
Wilson, Wells M.
Jones, Kenneth S.
Kneup, Frederick G.
Lynch, Leland C.
Maike, Arthur A.
Reed, Wesley R.
Washburn, Paul M.

Oregon
McCornack, R. C.
Russ, Robert S.

Pennsylvania
Craige, John E.
Lee, John M.

Rhode Island
Belinsky, Joseph

South Dakota
McGilvray, John H.

Tennessee
Bender, Jack D.

Texas
Couch, J. B.
Hander, Raymond T.
Schott, Francis J.
Thompson, Wm. M.

Virginia
Bowen, L. E., Jr.
Schachter, Jacob J.

Washington
Anderson, Walter A.
Younce, Ralph Ray

Wyoming
Humphreys, V. J.

Veterinary Officers Promoted

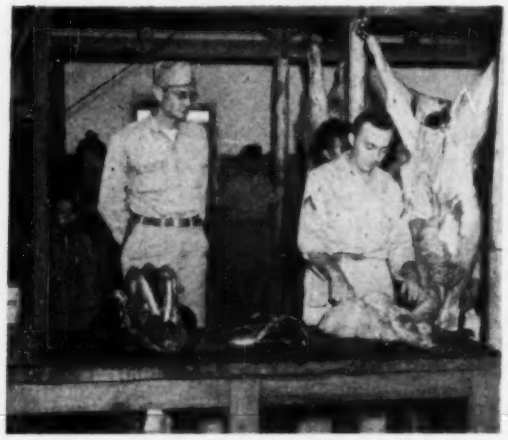
The office of the Surgeon General reports that Edward Chauncey Phipps (O.S.U., '34), Mayfield, Ky.; Albert Arthur Roby, Jr. (K.S.C., '34), Apopka, Fla.; and Marvin John Twiehaus (K.S.C., '33), Manhattan, Kan., have been promoted in rank from Major to Lieutenant Colonel.

Veterinary Officer Wins Bronze Star Medal

From the headquarters of the Eighth Army in Yokohama, Japan, comes word that Major Eugene W. Peck, V.C. (K.S.C., '33) has been awarded the Bronze Star Medal for meritorious achievement with military operations against the enemy in the Philippines and Japan.

U. S. Army Abattoirs in China

Abattoirs have been established by the U. S. Army in many parts of China, and are being supervised by army personnel in the American manner and for the purpose of supplying meat of good quality to American soldiers. Captain Thomas E. Utley, (Colo., '36), Maywood, Ill., is commanding officer of the 84th Veterinary Food Inspection Detachment which built and operated the abattoir at Chanyi, China, and is at present stationed at the Shanghai Municipal Abattoir.



—Photo by Carl R. Rammons

Pfc. Edward J. Oginski, of Chicago, inspecting beef viscera under the supervision of Capt. Thomas E. Utley, of Maywood, Ill.

—Photo by Carl R. Rammons

Sgt. Gerald R. Lombardo, of Chicago, weighing fresh beef prior to shipment to the mess halls.

The Army VC on meat is equivalent to the BAI "Inspected and Passed" stamp at home. It signifies that the animals furnishing the meat have been subjected to antemortem as well as postmortem inspection, and that the meat has been carefully handled after slaughter to prevent contamination. This has been accomplished through the use of Veterinary Corps officers and enlisted men with experience in the meat packing industry and on the live stock farms of America. These men have added to their civilian experience months and years of army schooling on meat and dairy inspection, and have proved their ability before being selected to perform these important tasks.

—Photo by Carl R. Rammons

T/5 Ralph D. Baird, of Findlay, Ohio, inspects the body glands of a beef carcass.

Carcasses are rejected daily because of pneumonia, tuberculosis, measles, anthrax, and other conditions, but those which pass inspection assure the military personnel a supply of meat in good sanitary condition. A continuous program of education among Chinese contractors has resulted in a gradual rise in the quality and the variety of meat available. Beef, pork, and chicken are seen regularly in the mess halls.

The American style of slaughtering, dressing, and dividing carcasses is used. An inspector is present at all stages of the work, and in doubtful cases, the Veterinary Corps officer is called to make the final inspection.

Were the world to revert to prewar status, there would be too many beef cattle in the United States for the good of the country, an expert economist declares.

An all-white-meat strain of chicken is being developed, according to a report coming from poultry breeders—which is not so cheery for the second-joint hound.


IN extending our very best wishes for 1946, we take this opportunity to renew our pledge to the veterinary profession — to produce the best biological products possible and to confine their distribution to those whom we consider most capable of using them — registered, graduate veterinarians.

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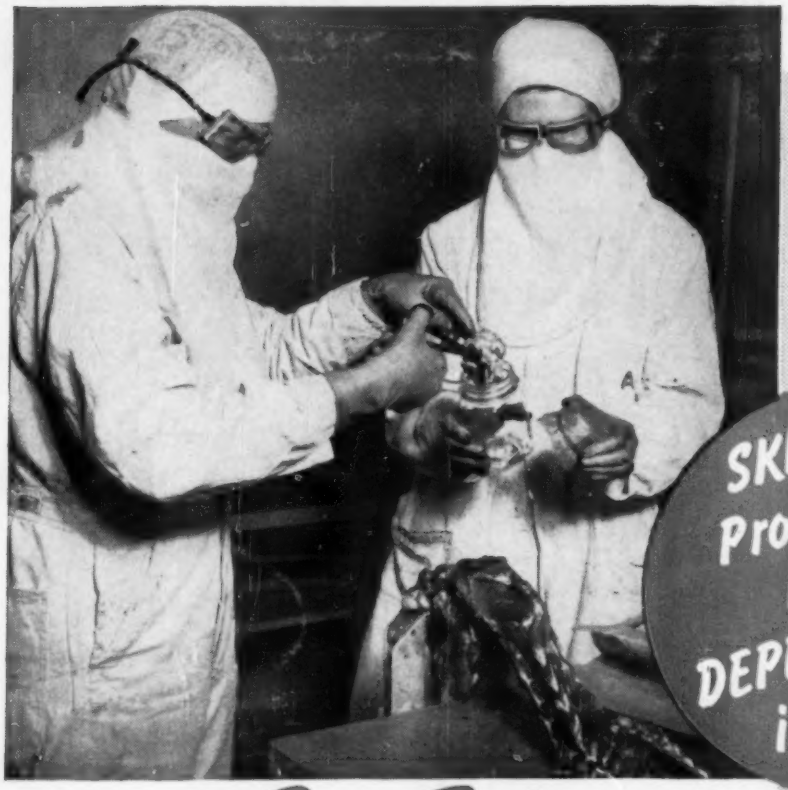

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